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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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November 6, 7, 8, 1958

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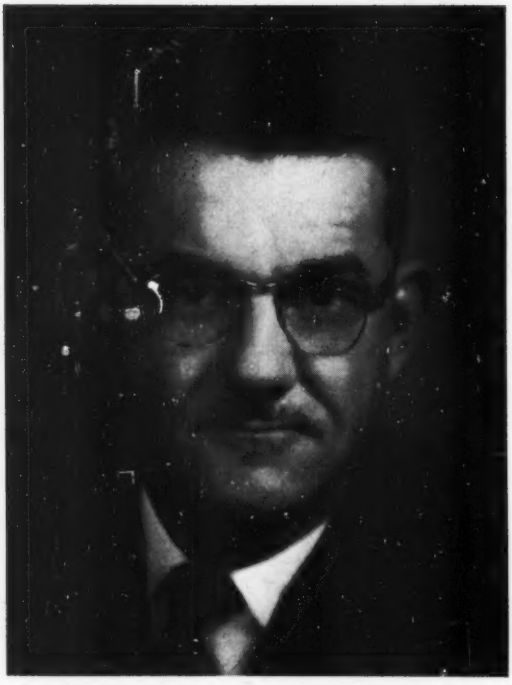

DONALD A. KERR, D.D.S., M.S.

President, American Academy of Periodontology, 1958

Dr. Donald A. Kerr of Ann Arbor, Michigan was installed as President of the American Academy of Periodontology at the 1957 meeting in Miami. Dr. Kerr has been very active, working on the Council and many of the committees of the Academy. He is a Fellow and past president of the American Academy of Oral Pathology, and a Diplomate of the American Board of Oral Pathology. He has served this group for many years as secretary treasurer.

He is also a member of the American Academy of Dental Medicine and a Diplomate of the American Board of Oral Medicine.

Dr. Kerr is Professor and Head of the Department of Oral Pathology and Periodontia at the University of Michigan School of Dentistry, as well as consultant to the Department of Oral Surgery of the University hospital and Veterans Hospital. He devotes a little time to private practice which is limited to periodontia and oral diagnosis. Dr. Kerr is also a Fellow of the American College of Dentists and a member of Omicron Kappa Upsilon, Sigma Xi, Phi Sigma, Phi Kappa Phi and Delta Sigma Delta.

His complete understanding of the problems which face the American Academy of Periodontology, as well as his knowledge of the specialty field itself, make it certain that he will be an effective and popular president.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-EIGHT

Effects of Various Diets on the Periodontal Structures of Hamsters*

by S. S. STAHL, S. C. MILLER AND E. D. GOLDSMITH, *New York, New York*

RECENT investigations have focused attention on the use of the syrian hamster in the study of periodontal disease. Periodontal lesions occurring in this animal have been described as localized interproximal destructions due to the impaction of hair and debris and a more generalized destruction associated with debris of powdered diets.[1-5] Various dietary as well endocrine factors have been investigated in order to observe the effects of these alterations on the character of the periodontal pathology. It was noted that females were more susceptible to periodontal disease. Protein deprivation also caused a slight increase in periodontal pathology.[6-9] The present investigation was undertaken in order to study the effects of a protein free diet on the periodontal structures of hamsters and to compare these changes with those brought about by food impactions resulting from the intake of a granular diet in these animals. In addition, the effects of replacement of a normal diet following deprivation was also observed.

MATERIAL AND METHODS

Seventy-four male syrian hamsters weighing an average of 106 Gms. at the beginning of the experiment were divided into three groups. Hamsters in group I, consisting of eight animals, were placed on a normal coarse Purina chow diet.* Four of these animals were sacrificed after sixty days, the remaining animals after one-hundred twenty days. Hamsters in group II, consisting of twenty-eight animals, were placed on a normal granular Purina chow diet.** Eighteen of these animals were sacrificed after sixty days, the remaining ten animals after one-hundred twenty days. Hamsters in group III, consisting of thirty-eight animals, were placed on a powdered protein free diet.*** After sixty days all of these animals had lost more than half of their starting weight and most of them appeared moribund. Three animals of this group acted alert and seemed to be less affected than the remainder of the group. These animals were placed on a normal coarse Purina chow diet and maintained for another sixty day period.

*This study was supported in part by a grant-in-aid D-269 from the National Institute for Dental Research of the National Institutes of Health, United States Public Health Service.

From the Murry and Leonie Guggenheim Foundation Institute for Dental Research, Department of Periodontia and Oral Medicine and Department of Histology, New York University College of Dentistry.

*Purina Lab Chow Checkers obtained from Ralston Purina Company, St. Louis, Missouri. **Purina Lab Chow Meal obtained from Ralston Purina Company, St. Louis, Missouri. ***Protein-free Diet Powder obtained from Nutritional Biochemical Company, Cleveland, Ohio.

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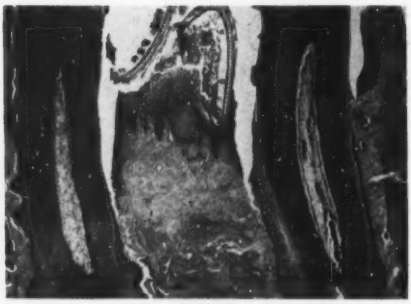

Fig. 1. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a granular diet over a two months period. Hematoxylin-eosin stain, Magn. 50X. Note the apical proliferation of the epithelial attachment as well as the food impaction in the interproximal gingiva.

All animals were fed ad libitum. At the end of the experimental period, the animals were sacrificed, the maxilla and mandible dissected out and prepared for histologic study. The tissues were fixed in formalin, cut at 7 micra and stained with hematoxylin-eosin.

HISTOLOGIC OBSERVATIONS:

Animals on a soft granular diet:

The striking change in this group was the reaction of the periodontal structures to food impactions. Due to the character of the food, hairs, kernels and other debris remained in the interproximal areas causing inflammation and apical proliferation of the epithelial attachment (figs. 1 and 2).

Of interest was the observation that in some animals keratinization of the interproximal epithelium was associated with lesser epithelial proliferation than that seen in animals where the epithelial changes consisted primarily of degenerative changes, such as intercellular and intracellular edema without attempts at keratinization (figs. 3 and 4). The alveolar crest in all animals showed remodeling with marked evidence of osteogenic activity being present.

ANIMALS ON A PROTEIN FREE DIET

The striking changes in this group of animals consisted primarily of an endosteal osteoporosis and a decrease in the connective tissue elements of the periodontal membrane.

Changes in the connective tissue of the periodontal membrane were noted after one week of dietary deprivation and appeared as a marked edema within the collagen bundles as well as a reduction in the number of fibroblasts present (fig. 5).

After one month, marked endosteal osteoporosis was seen in addition to the reduction of fibroblasts in the periodontal membrane. The collagen fibers appeared fibrillar and stained pale with hematoxylin-eosin. A reduction in remodeling activity of bone was also noted (figs. 6 and 7). Similar changes as observed after one month, were seen after two months of protein deprivation (fig. 8). It must of course be remembered, that the intake of food became more limited as the experimental time progressed, thus results described here may also reflect the reaction to partial starvation. Some epithelial proliferation of the attachment was noted in this group, however it was less extensive than seen in

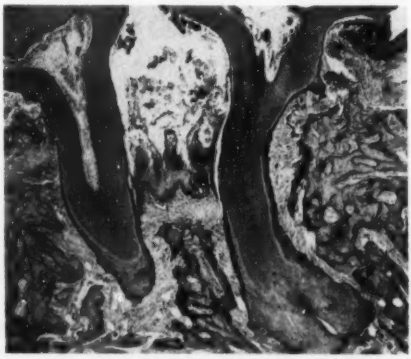

Fig. 2. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a normal granular diet over a three months period. Hematoxylin-eosin stain, Magn. 50X. Note the marked apical proliferation of the epithelial attachment.

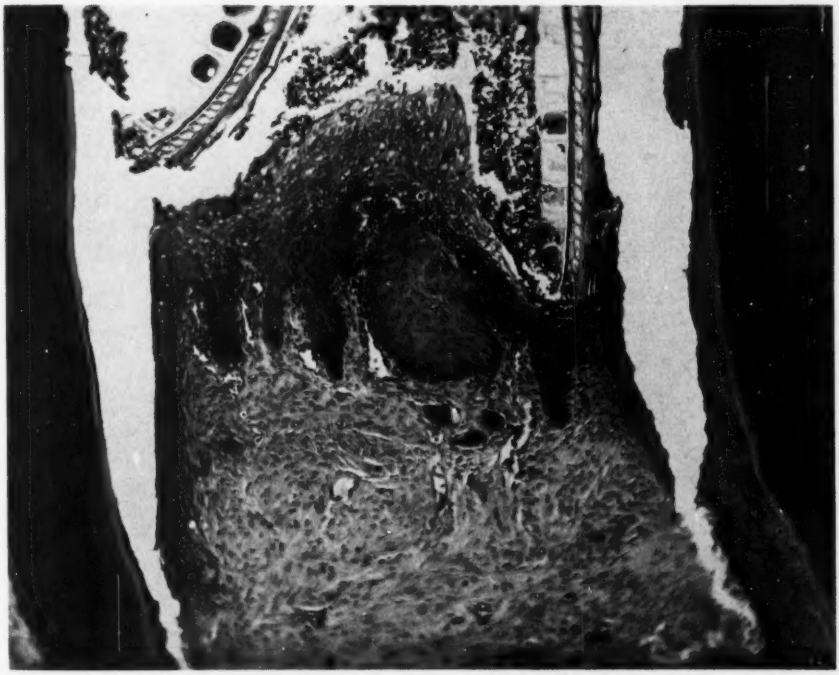

Fig. 3. Higher magnification of the interproximal gingiva shown in fig 1. Hematoxylin-eosin stain, Magn. 100X. Note the edema in the surface epithelium.

the animals on a soft diet and was usually found in areas of food impaction.

Reconstitution of a normal coarse diet after two months of deprivation led to an increased cellularity of the periodontal membrane and some decrease in the size of the marrow spaces. Increased proliferation of the epithelial attachment was noted in this group, however here too, food impaction was present in all instances. The extent of epithelial proliferation was not as severe as that seen in the group on a soft granular diet (fig. 9).

ANIMALS ON A NORMAL COARSE DIET

Animals in this group showed a lessened degree of food impaction, and epithelial proliferation was not extensive. This lesser impaction of food was seen after both two and four months periods and may be the

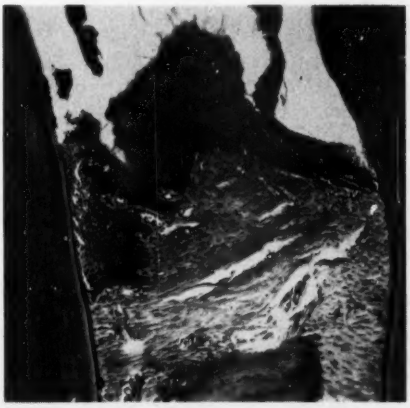

Fig. 4. Photomicrograph of an interproximal area between the first and second mandibular molars in a hamster fed a granular diet for two months. Hematoxylin-eosin stain, Magn. X100. Note the epithelial attachment near the cemento-enamel junction and the keratinized surface epithelium.

Fig 5. Photomicrograph of an interproximal area between the first and second mandibular molars of an animal fed a powdered protein free diet for one week. Hematoxylin-eosin stain, Magn. 100X. Note the edema within the transeptal fiber bundles.

reason for the rather limited epithelial proliferation (figs. 10 and 11).

The interproximal alveolar crest showed remodeling with marked osteogenic activity.

It was of interest to note that food impaction and periodontal pathology were more severe in the maxillary than in the mandibular teeth and were greatest between the first and second molars in all groups studied.

COMMENT

Our observations are in general agreement with the findings of previous investigators regarding the development of interproximal periodontal lesions due to the effects of debris and food impaction in the area.[10,11] The use of a protein free diet while causing osteoporosis as well as a de-

Fig. 6. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a powdered protein free diet for one month. Hematoxylin-eosin stain, Magn. 20X. Note the enlarged marrow spaces in the interradicular septi, and the reduction in cellularity of the periodontal membrane. The epithelial attachment is at the cemento-enamel junction level.

Fig. 7. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a powdered protein-free diet for one month. Hematoxylin-eosin stain, Magn. 50X. Note the enlarged marrow spaces in both the interdental and interradicular septi. The epithelial attachment is at the cemento-enamel junction level.

Fig. 8. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a powdered protein free diet over a two months period. Hematoxylin-eosin stain, Magn. 20X. Note the enlarged marrow spaces in the alveolar bone and the pale staining periodontal membrane. The epithelial attachment is at the level of the cemento-enamel junction.

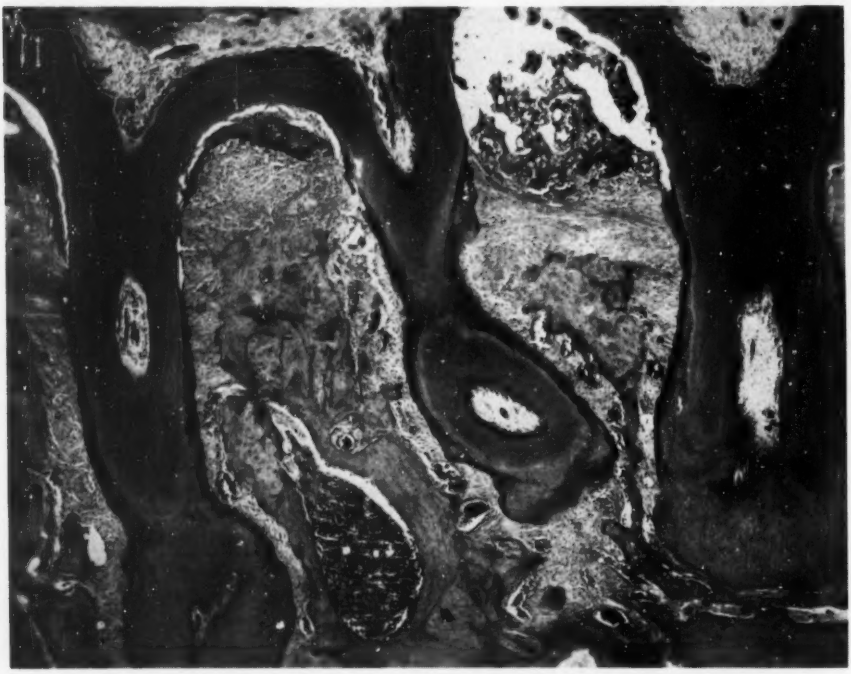

Fig. 9. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a powdered protein free diet for two months followed by a normal coarse diet for a subsequent two months period. Hematoxylin-eosin stain, Magn. 50X. Note the apical proliferation of the epithelial attachment below the cemento-enamel junction and the normal appearance of the marrow spaces.

crease in cellular activity of the periodontal membrane did not cause increased interproximal destruction with proliferation of the epithelial attachment. It is felt that the relative absence of periodontal pathology in the deprived animals may be due to the lack of food impaction resulting from the intake of a finely powdered diet. Mitchell and Johnson[12] too, observed no striking microscopic changes in their study of the periodontium of protein deprived hamsters.

The observations in protein deprived hamsters described above are in substantial agreement with the findings reported previously in other animals on a protein free diet; namely the increasing osteoporosis as well as the reduction in cellularity of the periodontal membrane.[13, 14, 15] This interference with growth and repair in itself apparently does not initiate downgrowth of the epithelial attachment. Local gingival irritants, on the other hand, in the present study food impaction, were associated in almost every animal with proliferation of the epithelial attachment. These findings are also in agreement with previous findings where gingival irritation in rats caused epithelial proliferation of the attachment. The superimposition of starvation or protein free diets in those animals led to increasingly severe periodontal lesions due to the inability of the animal to repair the injury.[16, 17] It appears then that some type of local gingival injury may cause the initiation of apical proliferation of the epithelial attachment, while systemic and occlusal factors play an important role in controlling the extent of the lesion following its initiation.[18] The reconstitution of a proper diet after chronic in-

Fig. 10. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a normal coarse diet over a two months period. Hematoxylin-eosin stain, Magn. 20X. Note the epithelial attachment at the cemento-enamel junction level.

jury is able to bring about healing of the lesion but is not able to restore the tissues to the level prior to injury.

SUMMARY AND CONCLUSIONS

Seventy-four young adult male hamsters weighing an average of 106 gms., at the beginning of the experiment were divided into three groups, Animals in Group I were placed on a normal purina chow checker diet. Animals in Group II were placed on a normal granular purina chow meal diet. Animals in Group III were placed on a powdered protein free diet.

A number of animals in each group were sacrificed after sixty days, the remainder after 120 days. Some animals in Group III received a normal diet for 60 days following 60 days of protein deprivation.

Histologic observation of the periodontal tissues showed that (1) protein deprivation caused endosteal osteoporosis with a reduction in cellularity of the periodontal membrane. The epithelial attachment level did not appear to be affected.

(2) The intake of a normal granular diet caused marked interproximal food impactions which were associated with downgrowth of the epithelial attachment, resorption of the interproximal alveolar crest and some evidence of inflammatory infiltrate in the area. The periodonal struc-

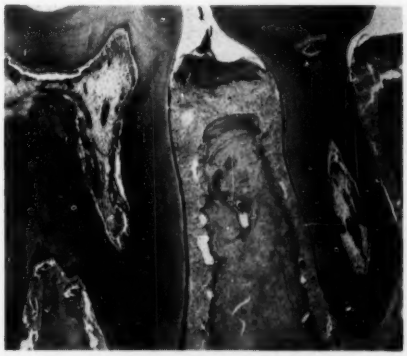

Fig. 11. Photomicrograph of a mesio-distal section of a mandibular first and second molar in a hamster fed a normal coarse diet over a four months period. Hematoxylin-eosin stain, Magn. 20X. Note the epithelial attachment at the cemento-enamel junction level.

tures of the maxillary molars showed more severe periodontal lesions than did the periodontium of the mandibular molars.

(3) Restitution of a normal diet following protein deprivation reduced the endosteal osteoporosis and increased the cellularity of the periodontal membrane.

The authors wish to acknowledge the technical assistance of Aileen Johansson, B.A., in the preparation of this study.

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MIDWEST CONFERENCE OF PERIODONTISTS ORGANIZED

During the Miami meeting of the American Academy of Periodontology, a small group of periodontists from the Midwest met and organized into "The Midwest Conference of Periodontists." The men responsible for this organization are Drs. Irving C. Stone and Jack C. Penticuff.

It is the plan of this group to arrange meetings periodically; at the organization meeting several committees were appointed in order to get the activity started. Dr. Clarke Chamberlain has been appointed as Honorary Chairman and the Advisory Board consists of Donovan W. Brown (Chairman), Maynard K. Hine, Donald A. Kerr and Robert C. Kesel.

Osseous Resection in Periodontal Surgery*

by CLIFFORD OCHSENBEIN, D.D.S., *Austin, Texas*

THE gingivectomy has for many years been the basic periodontal surgical procedure. Various techniques have been advanced for its performance. Its objective has been the elimination or reduction of abnormal sulcular depth, and in many cases this objective is achieved. There are, however, many other areas in which this objective has failed because of a regrowth of the remaining gingiva to its original preoperative level or nearly so. Analysis of reasons for this regrowth and the consequent reformation of pockets has revealed that the form which the gingiva takes is qualified to some degree by the architecture of the underlying bone.

If periodontal destruction consisted only of an apical migration of the bone, with no change in architectural topography, periodontal therapy would concern itself only with soft tissue problems. If this were true, most gingivectomies would be successful. The underlying bone remaining normal in form with interproximal cones well intact with knife edged margins, would lend itself to a long bevelled incision which would eliminate the pocket and create acceptable physiologic gingival contours. In other words, the postoperative result would be one of good architectural form but more apical on the root surfaces. Unfortunately this is not the typical pattern of bone loss. If one is dealing with incipient periodontal lesions, he may not be confronted with any real modifications in bone architecture, but since the periodontist must deal also in more advanced lesions, it is here that problems arise and must be met.

In health, bone and tissue are adjacent to each other and consistent with one another in form. Fig. 1 represents a normal, healthy gingiva. The margins are well adapted and thin, papilla is cone shaped and blends into the interdental groove. The physiologic contours possess self-cleaning qualities which are necessary for maintenance of health. Since this is a normal picture of health, all periodontal therapy should be directed toward this goal. This means that even in surgical management where the gingiva must of necessity be positioned further apically, the post operative result should still resemble this in form. Fig. 2 demonstrates the architectural pattern of bone usually found in health. This is the bone topography one would probably encounter if the soft tissue were removed from the bone and teeth of an individual such as that in Fig. 1. Notice that the form of both are quite similar.

In periodontal destruction, the architectural form is often modified or deformed. These deformities manifest themselves in bone as interproximal craters, heavy buccal or lingual ledges, infra-bony pockets, blunted interdental crests and numerous other bizarre patterns. This behavior is the exact opposite of that of soft tissue. The gingival tissue contour covering these deformities is often acceptable, or even enlarged; the difference in levels of bone and soft tissue are expressed in pocket depth. When the gingiva is excised in such cases it cannot adapt itself in healing to the deformed bony profile for any length of time. The inherent quality of soft tissue to revert to a scalloped pattern is responsible for a reformation of pocket depth. The hard and soft tissues are no longer consistent with, or adjacent to, each other.

The removal of bone in periodontal therapy is not new and had been advocated prior to Schluger's classic presentation in 1949.[1] The earlier work dealt with the removal of bone in the leveling of the infra-bony pocket. Schluger developed the concept and set forth basic principles of plastic repair, just as Goldman did in the gingivoplasty.[2] This concept deals with

*This paper was presented at the 1956 Meeting of the American Academy of Periodontology.

much more than bone removal for pocket elimination but enunciates the plastic principle of recontouring bone for the production of good physiologic architecture in all the involved tissues.[3] Pocket elimination is not enough, since one must also treat the deformity that occurs as a result of the disease process. By so doing, normal structure and function can be restored to the organ treated.

Kronfield stated in 1935 that bone tissue under periodontal pockets is inflamed but vital, and never necrotic.[4] It should be emphasized that any bone removed in cases described in this report was done for architectural reasons, and not because it is thought to be infected or necrotic. Healthy bone is removed in osseous resection, just as good healthy soft tissue is removed in the gingivectomy to achieve physiologic, self-cleansing qualities of the gingivo-marginal area.

The term "osseous resection" originally covered all phases of bone surgery in periodontal procedures. Recently, Friedman[5] divided this term into two divisions—osteoplasty and osteoectomy. *Osteoplasty* is, just as the term implies, a plastic recontouring of bone for physiologic architecture so that the gingiva may also have contours that are physiologic. This procedure of "festooning" the bone does not involve or sacrifice any of the supporting structure of the tooth. *Osteoectomy*, on the other hand, is the procedure of removing bone which is part of the supporting structure, in order to eliminate the pocket and also to create proper physiologic contours.

This writer feels that the above terms, "osteoplasty" and "osteoectomy," are useful terms in an academic sense. In doing an osteoectomy, one carries out all the principles of osteoplasty; the only difference being that some of the attachment apparatus is removed in osteoectomy. One of the purposes of this report will be to emphasize that most osseous surgery however, is osteoplasty and even in cases requiring extensive reshaping of the bone, the amount of supporting bone sacrificed is surprisingly small.

Fig. 1. Normal gingiva. The margins are well adapted, knife-edged, papilla is cone shaped and blends into the interdental groove. All periodontal therapy should be planned with these physiologic architectural objectives in mind.

The most common deformity to be found as a result of the periodontal lesion is the blunted interproximal crestal bone. It is common practice to find the pocket deeper interproximally than on labial or lingual surfaces,[6] and this may even be modified to state that it is not uncommon to see pockets on the interproximal and no pocket depth on labial or lingual aspects. Since the interproximal area is the chief site of the periodontal lesion and since the underlying bone in health is cone shaped from buccal to lingual, it is not difficult to realize that this cone of bone is resorbed, becomes blunted, and is therefore a common early deformity.

Blunting occurs on anterior and posterior interproximal bone, but most frequently in the posterior regions. Perhaps this could be explained by a comparison study of the anterior and posterior interdental architecture in health. The posterior cone is flatter and wider bucco-lingually in addition to having heavier cortical plates and can tolerate only minor changes before it becomes considerably modified. Fig. 3a will demonstrate a common deformity of this nature. Pocket depth of 4 m.m. was reduced on anterior teeth by gingivectomy. The interdental bone was found to be blunted and therefore limited the bevel that could be created by incision.

Fig. 2. Normal bone. Notice the structure and form and the similarity between it and the soft tissue architecture in Fig. 1.

A thick, blunted, interproximal papilla would be the result. Notice that the incision is little more than at right angles to the tooth in the area between the lateral incisor and canine. At the most apical extension of the bevel, one can insert an explorer into the tissue, readily probe bone, and determine that the bevel cannot be increased. Since the buccal margin of bone was not thick and an acceptable bevel could be obtained in the marginal areas, it was decided not to do a modified flap, but to utilize the diamond stones as originally advocated by Fox,[7] and reshape the blunted bone through the soft tissue, Fig. 3b. This is osteoplasty in its simplest form. The post-operative result, Fig. 3c, can be compared with the interdental area between the two central incisors, from an architectural standpoint, which has not been affected by periodontal disease. The gingival contour of the soft tissue is quite the same, the papilla is cone shaped, the marginal area is thin and blends into the interproximal area and groove. This promotes self-cleansing qualities in function and home-care is made easier for the patient.

The interproximal bony crater is a very common finding and is merely a more advanced lesion of the blunted interdental crest. It often occurs in the presence of heavy buccal and lingual cortical plates. Clinically, no pocket depth is noted at the line angles; but a probe makes a sharp drop as it progresses interproximally. Roentgenographically, the crater is often obscured because the heavy buccal and lingual plates are superimposed, giving a false image of the interproximal area. The soft tissue is often normal in appearance and intact along the heavy buccal and lingual plates of bone. Fig. 4a illustrates the topography of the lesion. The gingivectomy is limited to the buccal and lingual crests. The residual pocket depth plus the proliferation of the tissue coronal to the crests can only result in a regeneration of pocket depth. Such a result could be achieved with curettage. In order to eliminate the crater, one of the plates,

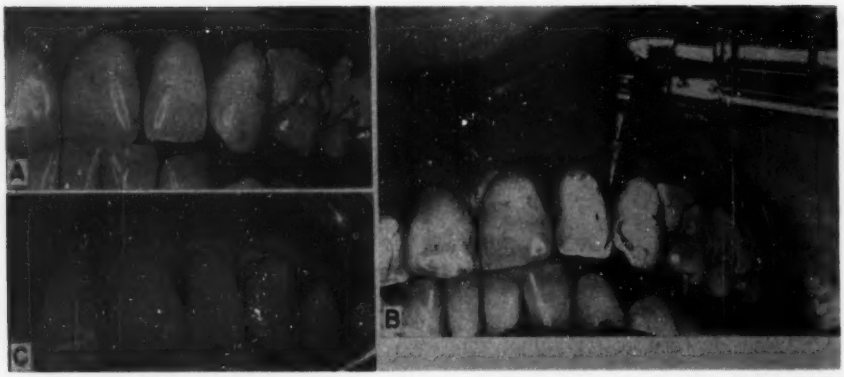

Fig. 3. 3a-Gingivectomy performed. Proper bevel was prevented by blunted interseptal bone. 3b-Osteoplasty thru the tissue with the aid of rotary abrasives. 3c-Post operative result of good interdental grooves and knife edged margins.

either buccal or lingual, must be reduced, Figs. 4b and c. It is usually the buccal plate because most patients find that it is easier to perform home maintenance from the buccal aspect and access is provided for the operative procedures.

After buccal or lingual plate has been reduced for crater elimination we are now confronted with the problem of creating physiologic form on the buccal or lingual side that was selected for recontouring. Fig. 4d shows the interproximal apical reduction performed, followed by Fig. 4e which shows the triangular removal of supporting bone on the two adjacent teeth. A gradual rise and fall of bone profile which is necessary for pocket elimination now exists. However, thick ledges of bone are still present in the interproximal areas. This presents the same problem as the blunted interdental crest. (See Fig. 3.) The procedure of festooning, Fig. 4f creates the vertical groove blending in with the papilla and adjacent areas for food passage.

The interproximal crater does have four hard walls for protection of a blood clot, however, reattachment in the crater type lesion has been extremely limited and one could hardly list it as a probable method of pocket elimination for the crater.

The isolated deep interproximal pocket should receive only a brief discussion due to the resemblance of the technique for its correction and that of the crater. This type of deformity is comparatively uncommon, while less precipitous supra-bony pockets are very common. Actually an isolated deep interproximal pocket is no more than a crater that has had both buccal and lingual plate reduction. The correction is the same procedure as that in Figs. 4e and 4f, for the crater. Tissue

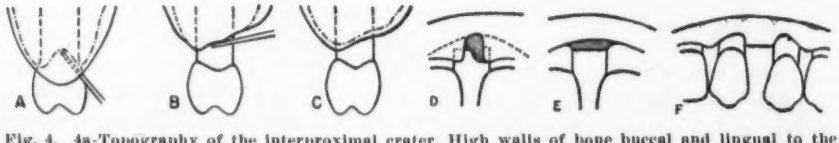

Fig. 4. 4a-Topography of the interproximal crater. High walls of bone buccal and lingual to the bottom of the pocket. 4b-Bone ramped to eliminate the saucer shaped deformity. 4c-Post operative result of the selective recession desired, viewed from buccal-lingual. 4d-Viewed from the side selected for recontouring, showing interproximal apical reduction. 4e-Demonstrates the removal of bone on the two adjacent teeth so that a proper rise and fall of bone profile is accomplished. 4f-Festooning principle, or creating the vertical groove, blending in with the papilla and adjacent areas for deflection of food. (After Schluger)

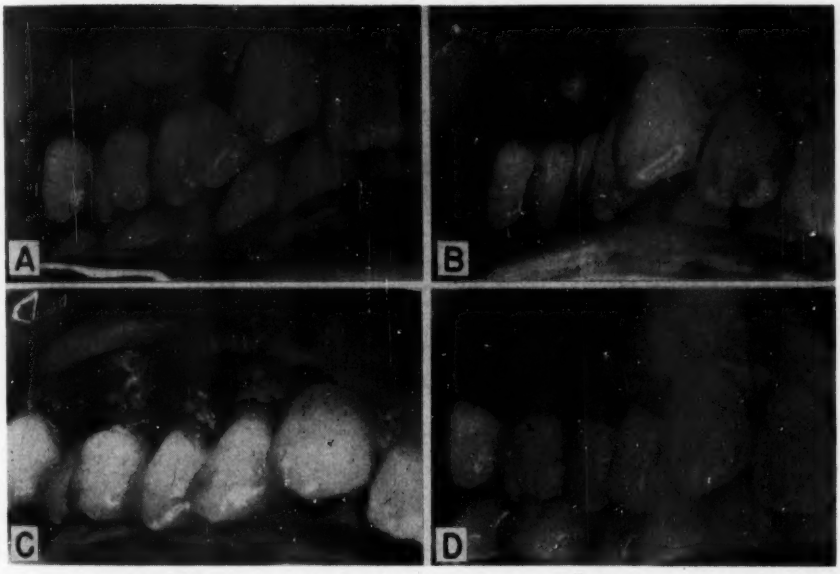

Fig. 5. 5a-Routine gingivectomy performed, incision made at right angles to the tooth due to thick bone ledge. 5b-Modified flap exposes the heavy thick plate of bone which would prevent the establishment of physiologic contours. 5c-Osteoplasty, or festooning of the ledges and interproximal blunted crests. No supporting bone was removed. 5d-Post operative result. Observe the vertical grooves and the papilla placed beneath the contact points.

will not adapt itself to a precipitous change in bone profile and it is only a matter of time until the pocket depth recurs to approximately its original depth. The gradual rise and fall of the underlying bone by sacrificing bone on the two adjacent teeth is apparently the best means we now have for treating this situation.

Bone loss on buccal and lingual surfaces, usually posteriors, frequently results in a loss of the knife-edged form and consequently, ledges and shallow, wide-mouthed, infra bony pockets are formed. This is particularly so if the buccal plate is heavy. In Fig. 5a, an incision was made which immediately eliminated the pocket. The heavy buccal plate present from canine to second molar became a very real obstacle and made it necessary to make an incision practically at right angles to the teeth. At this point one could expect no more than a flat interdental papilla and a heavy buccal ledge which would not deflect food. Long before this bone could reshape itself and thin out, the tissue would proliferate, pathology become re-established, and the surgery would have been in vain.[1] The bone topography becomes clearly evident when a modified flap is performed exposing the area, Fig. 5b. The buccal ledges and the interproximal blunted crests were festooned to conform to a physiologic pattern, Fig. 5c. This is a simple procedure and can be done in a fairly short time with practice. In Fig. 5d, the post-operative result is one of health; notice the interproximal, vertical grooves, the knife-edged gingiva and the papilla tucked in between the contact points. Again, this will maintain itself with the minimum of assistance from the patient.

The bifurcation and trifurcation involvement of molars present real problems to the periodontist and to the dentist performing restorative procedures. Many such involved teeth are in key positions for a prosthetic program and should the tooth be sacrificed, the entire treatment plan may be altered

Fig. 6. Lower molars with buccal bifurcation involvements. Hirschfeld silver points in place. There was no pocket depth on the buccal of the mesial or distal roots to the bifurcation. This is a typical pattern of bone loss and occurs frequently.

with a lowering of therapeutic objectives. Their roentgenographic and clinical detection are frequently overlooked and one must cultivate a thorough technique for the inspection of all bifurcations and trifurcations during the examination. The thin, worn, sharp curette is an excellent instrument for exploring the area as it can best follow the topography of the pocket. A close examination of bifurcation involvements on lower molars and buccal of upper molars reveals a very significant finding—the attachment level on the mesial and distal roots to the bifurcation involvement are nearly always at a much higher level. It is very common to probe a deep pocket involving the bifurcation and find no pocket depth to the mesial or distal. Fig. 6 is a roentgenogram of a lower first and second molar with Hirschfeld silver points[8] in place—the first molar having a 5 mm. pocket at the buccal bifurcation and the second molar with a 6 m.m. involvement. There was no pocket depth on the buccal of the mesial or distal roots. This particular patient presented with all lower molars involved in a similar pattern as described and yet was pocket-free in all other areas of the mouth. The tissue covering the bifurcations was in acceptable form with an exudate being the only symptom of disease.

The lower first and second molars in Fig. 7a have buccal bifurcation involvements. Shallow pockets were present on the mesial and distal roots with a much deeper pocket funneling into the interradicular area. On the second molar, bone was removed on mesial and distal roots to obtain similar levels of attachment. Osteoplasty was then performed on both teeth to produce a knife edge bone in marginal areas and to treat the bifurcation area as if it were an interproximal one. The latter part of this statement is worthy of explanation. The bone ledge apical to the bifurcation is ramped and hollowed out which produces a long bevel and vertical groove, Fig. 7b. This is quite like the interdental groove and is for the same purpose of providing an escape for food. The soft tissue at the bifurcation can now be recessed and behave as if it were an interdental area, Fig. 7c. This is of great importance and must be made so, surgically. A deep V-shaped gash would be the post-operative result of a

Fig. 7. 7a-Lower molars having buccal bifurcation involvements. Shallow pockets were present on the mesial and distal roots with deeper pockets funneling into the bifurcations. 7b-The bone apical to the bifurcation is hollowed out along with knife edging procedures in all areas. 7c-Post operative result. The bifurcation area has been recessed and treated similar to an interdental area. Food is deflected and home physio-therapy is easily performed.

Fig. 8. 8a-Pre operative picture demonstrating the soft tissue form overlying the bony deformities seen in 8b. 8b-Notice the aberrations in form, particularly the second molar. The standard gingivectomy could offer very little to this case. 8c-Post operative result. Attention is directed toward the papilla like pattern of soft tissue in the trifurcation area of the first molar. The bone has been ramped in the area of the second molar eliminating the funnel shaped deformity and the soft tissue reflects these plastic corrections.

simple gingivectomy for pocket reduction. This is an impossible profile for tissue to maintain and this factor coupled with the impingement of food on the thickened free margin, results in a heavy roll of tissue. It is only logical that the patient's access to the bifurcation is hampered and is a matter of time until the interdental stimulator must be pointed apically to reach the bifurcation. This is no less than a gouging effect into the tissue. An acute exacerbation may be the result.

The first and second molar area in Fig. 8a exhibits fairly scalloped soft tissue form; but in Fig. 8b we observe a sharp deviation from that of the overlying soft tissue. Note the bizarre pattern of the bone and particularly the deep funnel-shaped deformity on the second molar. Also notice the more extensive bone destruction on the mesial root of the first molar, than on the distal. Both molars have trifurcation in-

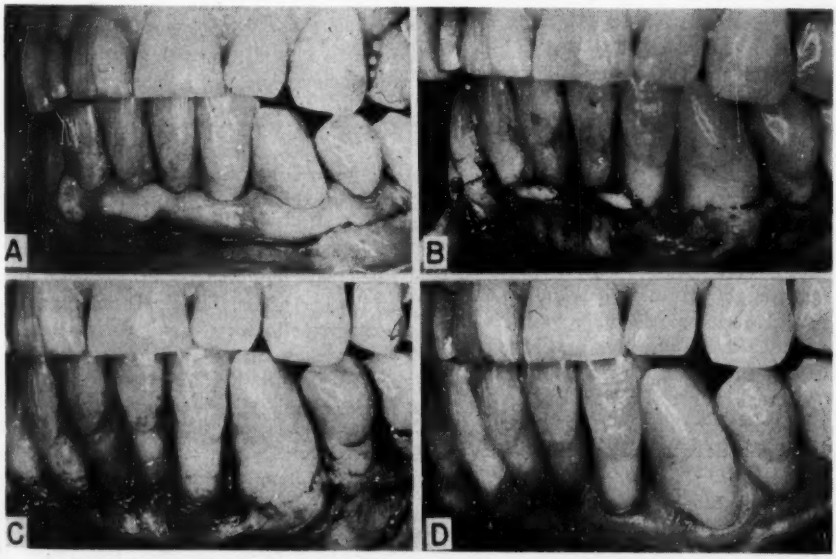

Fig. 9. 9a-Recording of pocket depth on gingiva and incision performed. 9b-Soft tissue removed and modified flap performed exposing the multiple craters and ledges on anterior teeth. 9c-Interproximal corrections were made and labial bone was sacrificed for good bone topography. 9d-Margins are well adapted, interproximal areas are cone shaped and blend into the interdental groove. Proper structure and function has been established.

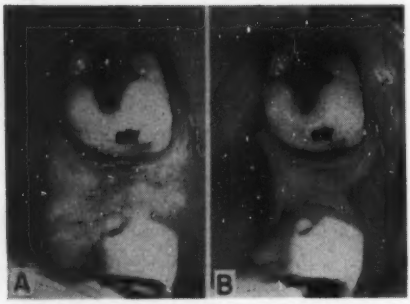

Fig. 10. 10a-The saddle area is broad and the restorative prescription calls for narrow bucco-lingual pontics. The saddle area must be narrowed to prevent the retention of food. 10b-Post operative result showing reduction of bucco-lingual width of the saddle. A satisfactory environment has been created for the narrow pontics.

volvements. The area was reshaped and bone was sacrificed on the distal root of the first molar. The lateral wall of bone making up the peculiar deformity on the second molar was reduced by osteoplasty. There was no supporting bone removed on the second molar. Fig. 8c is the post-operative result. Observe the first molar, with the papilla-like soft tissue in the trifurcation area resembling that of an interproximal one. Good deflecting contours are present, permitting proper food escape.

Most of the problems dealt with up to this point refer to posterior teeth. However, the anterior teeth demonstrate similar problems except that they are not so frequent or so bizarre. In Fig. 9a, pocket depths have been recorded on the gingiva and the gingivectomy incision carried out in the routine fashion. Fig. 9b, soft tissue is removed and the area presents multiple craters in conjunction with thick ledges or actually lip-like projections. After interproximal corrections of craters, high levels of bone remained on the labial surfaces of the incisors. Similar levels of bone were then produced in interproximal and labial areas by sacrificing labial bone, Fig. 9c. The post-operative result, Fig. 9d, is one of well adapted knife-edged margins, cone shaped interproximals, and good generalized contour. The marginal gingiva is now more apical to the cervical line of the tooth but a normal form with a near zero sulcus depth is present. Compare this final result in regard to form with Fig. 1 of a normal healthy gingiva.

Premolar and molar saddle areas can be extremely broad precluding a satisfactory environment for the pontic in restorative procedure. The distal abutment for the fixed restoration, occasionally presents a broad bucco-lingual saddle area upon which food can be retained after passing over the pontic. This is difficult for patient maintenance and is not in keeping with our principles of physiologic contours for deflecting food. Many dentists subscribe to the narrowing of bucco-lingual diameters to both abutments and pontics. This would aggravate the problem of food retention, particularly if the saddle areas were broad.

Fig. 10a will demonstrate the heavy broad saddle area. The second molar has a buccal bifurcation involvement and as this tooth is to be used for an abutment, it must receive as much assistance as possible. The crowns and pontics were to be narrowed considerably and the broad platform-like saddle area must be reduced bucco-lingually also, if continuity is carried out in the treatment plan, as shown in Fig. 10b.

The multiple interproximal crater is, without a doubt, the most difficult of all osseous surgery. The operator's judgment is constantly being challenged and it is also here that compromises in post-operative results can occur. The final result is often not correct in form, particularly in molar areas because buccal and lingual plates extend occlusalward from interproximal corrections. A leveling of buccal and lingual plates to correspond with the interproximal corrections would open up bifurcations and trifurcations. This would be radical surgery and certainly contraindicated.

In these areas between interproximal craters, the choice inevitably arises whether to sacrifice the tooth or teeth in question, or to perform rather extensive or difficult

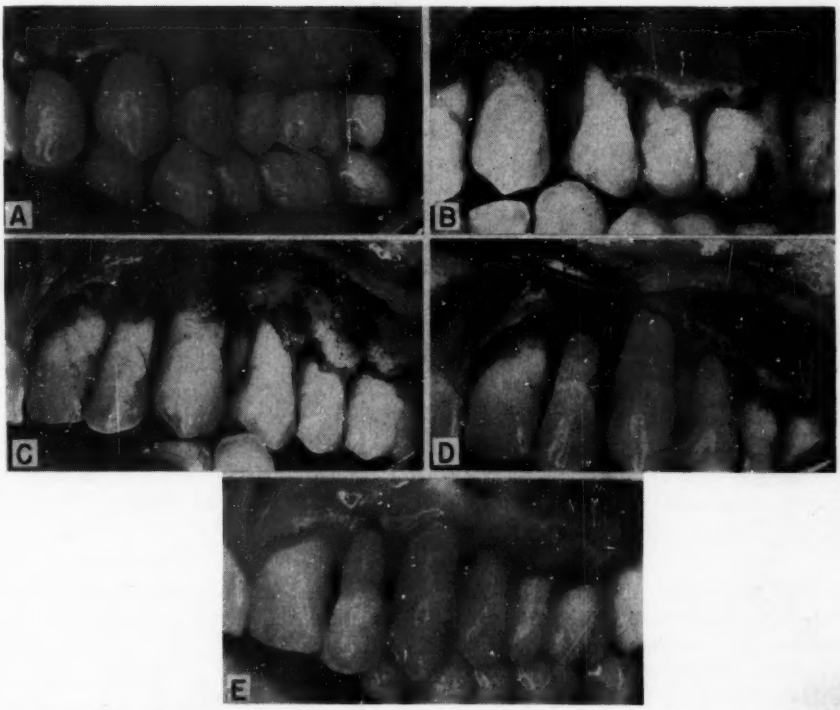

Fig. 11. 11a-Pocket depths interproximally range from 7 to 10 m.m. with shallow pockets on the buccal or labial and lingual. This pattern of pocket depth readily indicates multiple interproximal craters. 11b-Modified flap lifts the veil of soft tissue and exhibits the deformed bone pattern. Compare this with the normal bone form in Fig. 2. 11c-Interproximal correction, reducing the labial or buccal wall of the crater. Reversed or inverted pattern now needs further correction. 11d-Osteoectomy, or sacrificing labial or buccal bone to create acceptable structure and form. 11e-Post operative result is not ideal but acceptable. There is much selective recession but the architectural form is physiologic with the exception of the inverted pattern between the molars.

bone recontouring. The answer to this question lies with the resourcefulness of the operator, the critical importance of the teeth to the restorative treatment plan and all the other factors which call upon the operator's judgment.

Fig. 11a illustrates a hyperplastic and edematous area around anterior teeth, the posterior soft tissue is prominent due to a heavy buccal plate; but the architectural pattern could be considered fair. Interproximal pocket depth ranges from seven to ten millimeters, with very shallow or no pocket depth on the labial, buccal and lingual of anterior and bicuspid teeth.

A gingivectomy was performed along with a modified flap operation. Fig. 11b shows the veil of soft tissue lifted and the underlying pattern of bone exposed. Deep bone craters are in evidence, the first premolar has an early bifurcation involvement, buccal ledge is very prominent on second premolar and first molar. A buccal trifurcation involvement exists on the first molar with a deep crater between it and the second molar. The first molar also has a trifurcation involvement on the mesial between mesio-buccal and lingual roots. The second molar has a distal trifurcation involvement. The deep pockets on the anterior teeth and the possibility of a poor esthetic pattern after pocket elimination must also receive consideration.

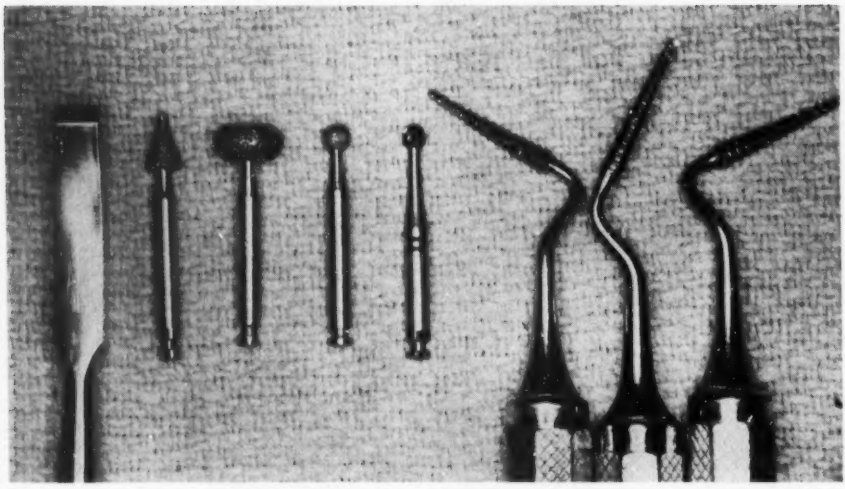

Fig. 12. Armamentarium. Left to right; bone chisel, Fox diamond stones, Carbide round bur, Buck files.

Fig. 11c demonstrates interproximal correction, making buccal and labial plates more coronal than interproximal areas. Fig. 11d shows the sacrificing of labial and buccal bone to create a fairly acceptable architectural form. If this procedure had not been performed, a generalized reversed pattern of soft tissue form would have resulted. The crater was eliminated between the molars but similar levels of bone could not be carried out, or even gradualized to the adjacent teeth as this would have required the sacrifice of too much buccal bone on the molars.

Fig. 11e is the post-operative result which reveals an acceptable gingiva form, with the exception of the reversed pattern between the molars. This compromise was anticipated, as stated previously. There is much selective recession but the gingival contour is within physiologic limits for long term maintenance in function.

The anterior teeth were later reshaped for esthetics but this reshaping was purposely postponed so that the amount of selective recession could be observed. (An important point to remember concerning anterior teeth with deep pockets—if good architectural form is produced in hard and soft tissues, the esthetic problem is reduced considerably.)

TECHNIQUE

The gingivectomy performed by the writer is the modified Black Operation. Briefly, it consists of measuring the pocket and recording this depth on the gingiva with a puncture mark. These puncture points are then connected by an incision made on a long bevel and the soft tissue forming the lateral wall of the pocket is removed. The modified flap is the next procedure for gaining access to the bone. This is by far the most acceptable method and is therefore used on practically all cases. A periosteal elevator may be used to detach the soft tissue from the bone. The writer uses a curette which is reversed and with this instrument a flap can be laid in a few seconds. The flap usually need not extend apically more than four or five millimeters, depending on the circumstances.

The round carbide bur will perform actually better than the coarse diamond stones, particularly after the diamond

stones have been used several times. However, the bur has its limitations and cannot be used when supporting bone is removed or beveled. In both instances the operator is likely to damage the tooth. This phase can best be performed with hand instruments. The chisel, Fig. 12 is very useful in the armamentarium and is utilized where marginal bone is removed. If the marginal bone is thick, it should be thinned down first with diamond stones and then marginal reduction is performed with greater ease.

The modified Buck file, is an excellent instrument for interproximal correction of the crater. In cases where the buccal plate is extremely heavy, the festooning of the interproximal should be performed first, then the interproximal apical reduction for crater elimination is a simple matter.

The short flap is debrided and without the aid of sutures is adapted to the exposed bone. If excess is evident, the flap can be trimmed with scissors. If the flap will not cover the bony field, due to retraction, adhesive foil can be placed over the area, followed by the preferred surgical dressing application.

When interdental grooves are accomplished in the festooning procedure of osteoplasty for interproximal areas, occasionally the gingiva will not adapt itself to the newly contoured areas. The tissue is taut over the grooves and the void between tissue and bone fills in with a blood clot. The end result may not be proper tissue form and therefore defeat our purpose. Vertical incisions of about 5 m.m. in the soft tissue over the recessed interproximal areas will permit the tissue to drape into, and adapt itself to the existing bone contour.

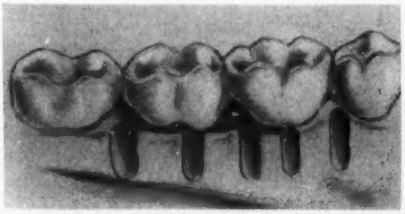

Fig. 13. Segmentation reduction. This procedure is used particularly on the lingual of the lower posterior teeth where heavy, thick ledges of bone or exostosis are present.

Extremely heavy buccal or lingual plates and exostosis in areas involved with pocket depth frequently require much bone reshaping. Even though rotary abrasives and burs are utilized, the procedure can be time-consuming, trauma-producing, and invite post-operative complications. The lingual of the lower is a very common example as it frequently presents a broad shelf of bone; the molar area often having both the shelf and a wide, shallow, infrabony type deformity in conjunction. This is a good area to demonstrate because it deals with a bulk of bone to be reshaped, it is in an area where access can be difficult, and where post-operative problems are always a factor to be considered.

After gingivectomy has removed the excess soft tissue, a modified flap is performed. A bulk of bone is immediately visible to the operator. Extensive tissue retraction is contra-indicated and seldom necessary. A carbide bur is used to make vertical sections in the bone, Fig. 13. As stated, very little retraction is necessary because the groove is started coronally, approximately 1 m.m. from the tooth. The groove is extended apically and lingually, the exact bevel not concerning the operator at this point. As many sections are made as are needed; grooves are interproximal and interradicular. It is then a simple matter to engage each section separately with a sharp chisel and with a slight tap, the offending bone is removed. At this point the lateral soft tissue collapses now that gross bone reduction has been accomplished. This procedure has been termed "segmentation reduction."

The large rotary abrasive is used in a contra-angle with the motor reversed so that the soft tissue will be forced back for good visibility. This also prevents unnecessary tissue laceration. With little effort the area can be finished down to proper contour.

SUMMARY

Bony modifications or deformities resulting from periodontal disease, preclude the possibility of hard and soft tissue being consistent with each other following therapy, unless the aberrations in form are corrected. The removal of large or small amounts of bone is incidental. The problem of creating deflecting contours for proper form and function is the primary concern.

Various types of bone deformities have been presented with possible solutions for their correction.

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BACK ISSUES NEEDED

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12	1	26	1
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The Lateral Periodontal Cyst

by SAMUEL M. STANDISH, D.D.S., M.S. AND WILLIAM G. SHAFER, D.D.S., M.S.,
Indianapolis, Indiana

THE lateral periodontal cyst is an uncommon lesion which is classified as an odontogenic cyst developing along a lateral root surface. The standard textbooks of oral pathology present the classical diagrams showing the mode of development of the odontogenic cysts and suggest that the lateral periodontal cyst has sometimes been confused with the lateral dentigerous cyst. Thoma[1] points out the rarity of the lateral periodontal cyst and the difficulty which may be encountered in differentiating this from the lateral dentigerous cyst. A case is illustrated by Stones[2] and identified as a lateral periodontal cyst situated adjacent to the root of an erupted mandibular third molar, but this cannot be judged an unequivocal case. Bernier[3] suggests that when the lateral follicular (dentigerous) cyst develops after enamel formation, expansion of the cyst may be so slow that there is no interference with tooth eruption and eventually the cyst will come to lie in apposition to the lateral surface of the tooth root. A case diagnosed as a paradental cyst, has been reported by Mezrow,[4] which was noted by Cross[5] to represent very likely a lateral periodontal cyst. Cross has presented a case of his own and suggested that these lesions might eventually manifest themselves clinically as lateral periodontal abscesses and thus fail to be accurately identified. Robinson, Koch and Kolas,[6] discussing the lateral periodontal cyst, state that this lesion usually develops within a lateral abscess or from pulpal infection extending through an accessory lateral root opening. They have illustrated such a case associated with a maxillary first bicuspid with an inadequate root canal filling. An excellent illustration of a lateral periodontal cyst attached to a maxillary third molar is reproduced in the Color Atlas of Oral Pathology.[7] The authors reported that such cysts frequently arise from the cell rests of Malassez and occur most commonly associated with vital, partially erupted third molars as a result of chronic pericoronal inflammation. They also indicate the possibility of a relationship to a lateral accessory pulp canal.

The high incidence of potential and actual developmental cysts about the jaws has been demonstrated by Scott[8] in a detailed histologic study of embryologic and fetal material. Kreshover[9] recently reported the occurrence of 65 single and multiple cysts of the gingiva in autopsy material from 17 infants. He stated that these cysts arose from cell remnants of the dental lamina and that certain of these cysts persist into adult life as so-called gingival cysts. Orban,[10] Ritchey and Orban,[11] Hodson,[12] Bhaskar and Laskin[13] and Kennedy[14] have reported cases of gingival cysts. There is no uniform agreement concerning the origin of these cysts but most writers generally believe that they arise either from traumatized and implanted oral epithelium,[10,11,12,13] from developmental remnants[9,11,12] or through cystic degeneration of surface epithelial clumps or pegs.[11,12]

Those cysts in adults which have been described in the literature as either gingival or lateral periodontal cysts have been distributed as follows: 7 in the mandibular cuspid-bicuspid region, 1 in the mandibular lateral incisor-cuspid area, 1 each in the maxillary central-lateral incisor, lateral incisor-cuspid, bicuspid-molar and third molar areas. The location of 1 cyst was undetermined. This interesting predominance for occurrence in the anterior segment of the mandible is further emphasized by the 5 additional cases of lateral periodontal cysts to be presented here.

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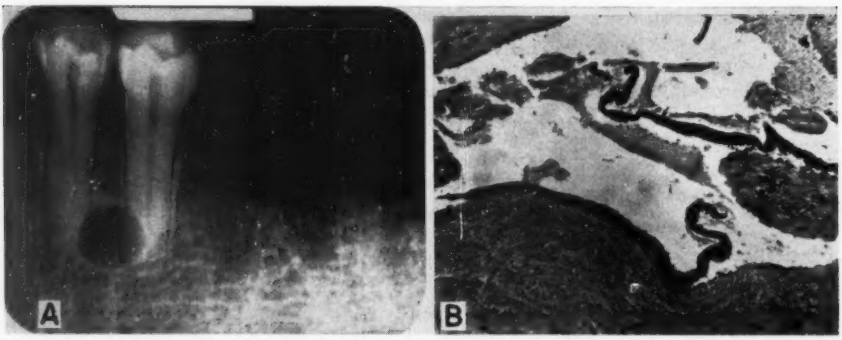

Fig. 1, Case 1. A) The intra-oral roentgenogram demonstrates the cystic area continuous with the periodontal membrane of the mandibular right first bicuspid. B) The high power photomicrograph shows the epithelial lined cavity and the absence of inflammation.

CASE HISTORIES

Case 1—A 64 year old negro male was seen at the Indiana University School of Dentistry for routine extraction of all remaining teeth and the construction of dentures. This patient had no specific complaint and the past medical history was negative. Intra-oral examination revealed fair oral hygiene, numerous missing teeth and a moderate periodontitis. Intra-oral roentgenograms revealed a mild degree of alveolar crest resorption about the remaining natural teeth. Near the apical third of the lower right first and second bicuspid teeth, a well-demarcated radiolucent area was noted (Fig. 1A). This area was located between the roots of these teeth in intimate association with the first bicuspid and was surrounded by a thin, clearly defined, radiopaque border. These particular teeth showed no carious lesions and there was no pulpal involvement. This radiolucent area did not appear to communicate with the gingival crevice. The cystic area was enucleated and submitted for histologic examination.

Histologic examination revealed a dense, fibrous connective tissue wall enclosing a central lumen and lined by a layer of stratified squamous epithelium (Fig. 1B). This epithelium was thin and showed no rete peg formation or proliferation into the connective tissue. There was no inflammatory cell infiltration of the connective tissue.

Case 2—A 52 year old negro female was seen at Indiana University School of Dentistry for examination and roentgenograms of a fractured maxillary right lateral incisor tooth. The patient had several missing posterior teeth. The periodontal tissues were in good condition and full mouth roentgenograms showed essentially normal supporting bone. However, in the mandibular left lateral incisor and cuspid region a large radiolucent area surrounded by a thin radiopaque border was noted between the roots of these teeth (Fig. 2A). The lesion appeared to involve the periodontal membrane space of the cuspid. Previous medical history included treatment for high blood pressure, an undetermined heart condition and a bilateral mandibular fracture suffered 15 years previously. At surgery, the cystic area was easily enucleated.

Histologic examination revealed a wall of connective tissue comprised of loosely arranged collagen fibers and stratified squamous epithelium surrounding a central lumen (Figs. 2B, C). The epithelial cells in some areas occurred in sheets and presented a distinctive appearance with a clear cytoplasm and no semblance of intercellular bridges. The cells are quite unlike those seen in the other more common periodontal cysts.

Case 3—A middle-aged white female was referred to an oral surgeon by her dentist for treatment of a bony defect noted between the roots of the mandibular left cuspid and first bicuspid teeth (Fig. 3A). The cuspid tooth with an attached soft tissue mass was extracted and submitted for histologic examination. No other pertinent information was available.

Histologically, the specimen consisted of a fibrous mass which was continuous with the periodontal membrane of the tooth and contained a central lumen lined by a layer of stratified squamous epithelium (Figs. 3B, C). Occasional epithelial rosettes were noted. There was infiltration of the connective tissue by lymphocytes and plasma cells in considerable numbers.

Case 4—A 59 year old negro female visited Indiana University School of Dentistry for extraction of 5 remaining mandibular teeth and the construction of dentures. The maxillary arch was edentulous and presented no pertinent features. Intra-oral roentgenograms revealed a well-defined radiolucent area about midway along the distal surface of the root of the mandibular right cuspid (Fig. 4A). The mandibular right first bicuspid had been extracted several years previously. This area was asymptomatic

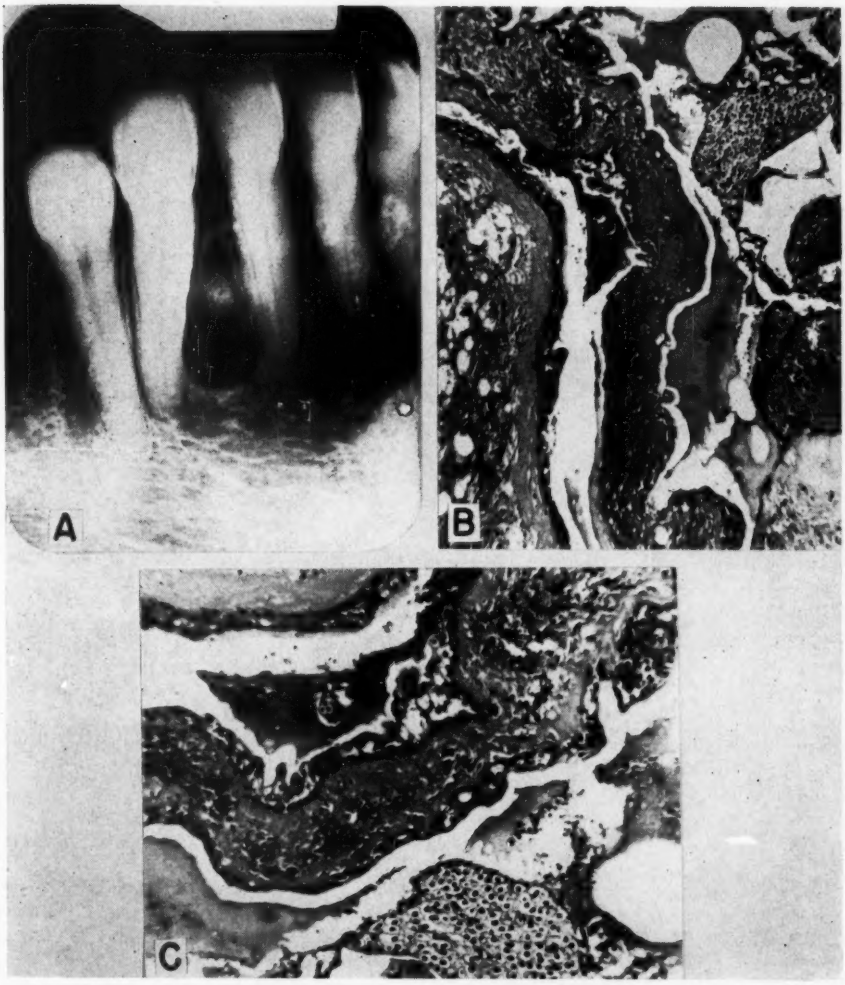

Fig. 2, Case 2. A) The intra-oral roentgenogram shows a cystic space intimately associated with the mandibular left cuspid tooth. B) The low power photomicrograph shows the cyst epithelium. C) The high power photomicrograph shows the lining epithelium and the tortuous nature of the cystic cavity.

and the cuspid tooth presented no carious lesions or restorations. Intact bone overlaid this area and no communication with the gingival crevice could be found. The cuspid tooth with the attached mass was removed in toto and submitted for histologic examination.

Histologic section revealed the connective tissue wall of the mass to be continuous with the periodontal membrane and to contain an epithelium lined lumen (Fig. 4B). There was no inflammatory cell infiltration of the connective tissue.

Case 5—A 41 year old negro male presented at Indiana University School of Dentistry for treatment of a tender, firm swelling on the buccal aspect of the mandibular left cuspid and first bicuspid area, approximately 1 cm. below the gingival crest. Previous medical history was negative and the patient presented a good dentition and periodontal structures. A defect in the bone could not be identified with intra-oral roentgenograms (Fig. 5A). At surgery, a sac-like structure was easily enucleated from a smooth, dished out depression in the underlying

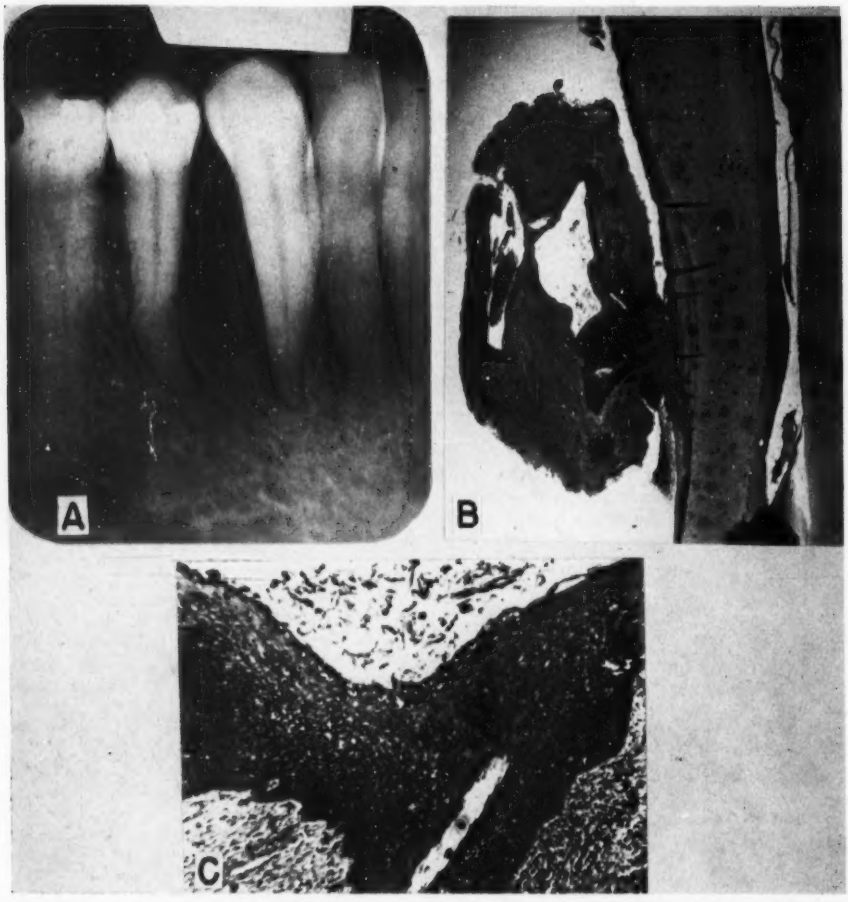

Fig. 3, Case 3. A) The intra-oral roentgenogram demonstrates a bony defect in the left mandibular cuspid-bicuspid area. B) The low power photomicrograph shows the cystic mass attached to the mandibular left cuspid. The connective tissue appears continuous with the periodontal membrane of the tooth. C) The high power photomicrograph demonstrates the proliferating cyst epithelium.

bone which the surgeon reported to be adjacent to the bicuspid.

Histologic section revealed a connective tissue mass with several tortuous epithelium lined lumina (Figs. 3B, C).

DISCUSSION

Evaluation of the pertinent literature combined with the five cases presented here emphasizes a number of important features concerning lateral periodontal and gingival cysts.

Eight of 14 lateral periodontal or gingival cysts occurring in adults, and which are reported in the literature in detail, were found in the general area of the mandibular cuspids and bicuspids. Two of these 8 cysts were classed as lateral periodontal (or paradental) cysts and six were diagnosed as gingival cysts. Three additional cases reported as gingival cysts were located in the maxillary anterior region and in 2 others the location was not stated. All 5 cysts reported here, and diagnosed as

lateral periodontal cysts, occurred in the mandibular cuspid and bicuspid regions. This remarkable predilection for the anterior segment of the mandible would seem to be more than a chance occurrence. The fact that 4 of the 5 cases reported here were in negroes may also be of significance although this cannot be explained.

The possibility that the lateral periodontal cyst actually represents a lateral dentigerous cyst left behind an erupting tooth must be considered.[3] If such were the case in this series, then obviously these cysts must have been present in these patients for a minimum of 25 to 54 years, assuming that they originated even as late as 10 years of age. It might be pointed out that a dentigerous cyst in the anterior portion of the mandible is an extremely uncommon occurrence. The lateral dentigerous cyst which occurs in the mandibular third molar region ordinarily presents little difficulty in diagnosis.

The high incidence of cystic lesions in the gingiva and about the jaws of children[8, 9] has been well established and such a cyst could conceivably erode bone and come to lie in contact with the roots of more than one tooth.[13] However, each of the cysts reported here was demonstrated at surgery, or histologically, to be intimately associated with the periodontal membrane of a single tooth.

The traumatic implantation of oral epithelium has been proposed by a number of investigators[10, 11, 12, 13] as a factor in the pathogenesis of the gingival cyst. The role of trauma in the production of the lateral periodontal cyst is particularly nebulous. The present series of cases gave no indication of trauma except for case 2 in which a bilateral mandibular fracture was sustained 15 years previously. No further details could be obtained regarding the exact location of the fracture.

Robinson, Koch and Kolas[6] indicate that the lateral periodontal cyst forms "along the root surface, usually, within a lateral

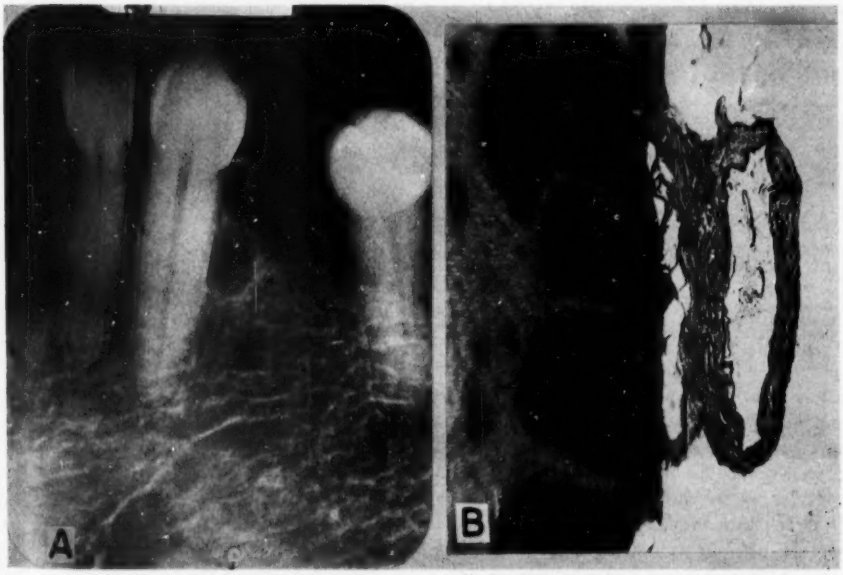

Fig. 4, Case 4. A) The intra-oral roentgenogram shows the bony defect adjacent to the mandibular right cuspid tooth. B) The photomicrograph demonstrates the attached cyst.

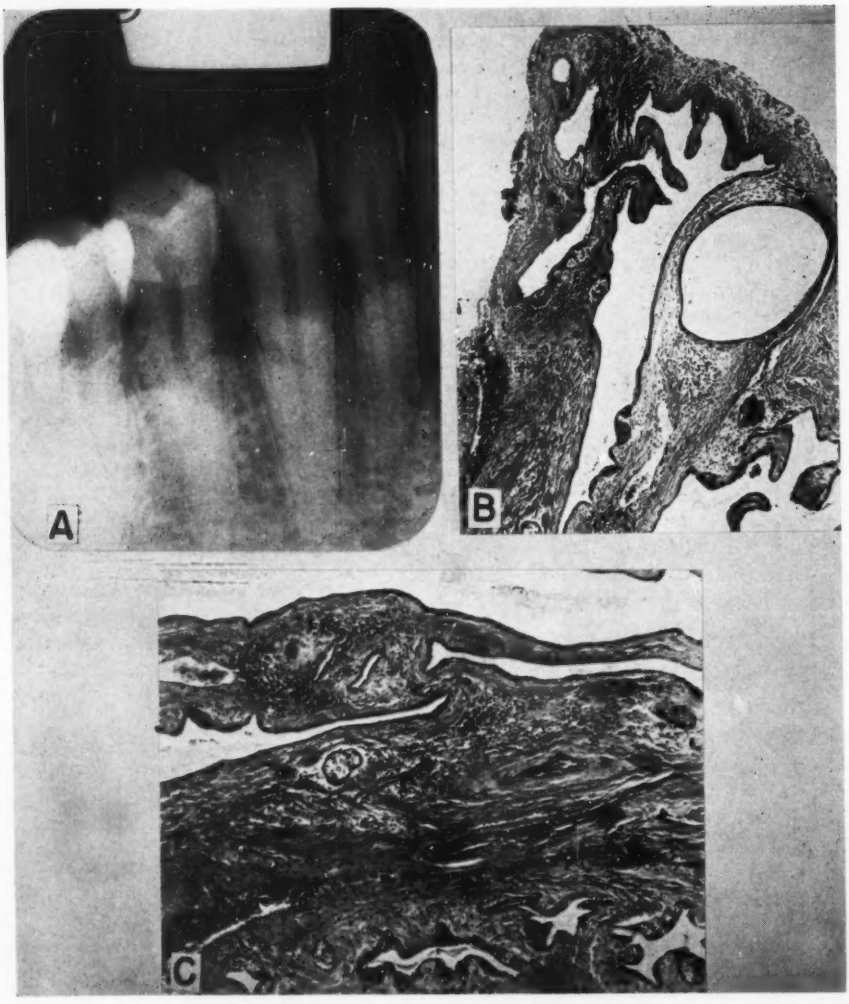

Fig. 5, Case 5. A) The intra-oral roentgenograms failed to demonstrate the superimposed lesion. The surgeon reported the cyst was in bone and in apposition to the periodontal membrane of the bicuspid tooth. B) The low power photomicrograph shows apparent microcyst formation adjacent to the main cyst. C) At higher magnification the lining epithelium as well as adjacent microcysts may be seen.

abscess, or from pulpal infection extended through an accessory root opening." It is our belief that the term "lateral periodontal cyst" should be used to indicate all cysts developing in the anatomic region of the lateral periodontal membrane but, because of the obvious varied etiology of this lesion, the term should be qualified to indicate whether the cyst has arisen as a result of pulp infection, infection through the gingival sulcus or through an idiopathic stimulation of cell rests.

The high incidence of epithelial cell rests in the periodontal membrane is well known and these are undoubtedly the source of the epithelium in the lateral periodontal cyst. However, the enigma of the stimulus re-

sponsible for the proliferation of these epithelial cells to form the lateral periodontal cyst in the absence of inflammation remains unsolved. Case 3, in our series, showed typical epithelial cell rests and cases 2 and 5 demonstrated proliferating clumps of epithelial cells apparently arising independent of and adjacent to the cyst. While complete serial sectioning of the lesions was not carried out, low power magnification of certain of these cases suggests that microcysts are forming adjacent to the cyst rather than as diverticula of the parent cyst (Figs. 2B, 5B).

The lateral periodontal cyst is a distinct clinical entity although of varied etiology which, in many cases, will be extremely difficult or impossible to differentiate from the gingival cyst, the atypical apical periodontal cyst or from complicated lateral periodontal abscesses.

SUMMARY

1. Five cases of the lateral periodontal cyst have been presented, all of which occurred in the mandibular cuspid-bicuspid area.

2. The lateral periodontal cyst arises from epithelial cell rests commonly found in the periodontal membrane although in this series the stimulus for the epithelial proliferation was not obvious.

3. Points of differentiation of the lateral periodontal cyst from the gingival cyst, atypical apical periodontal cyst and complicated lateral abscess have been emphasized.

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Histochemical Distribution of Acid Phosphatase in Human Gingiva

by Rómulo L. Cabrini, M.D.* and Fermín A. Carranza, Jr., D.D.S.,
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SINCE Gomori described a histochemical technic for the detection of phosphatases[4,5] many reports have appeared in the literature dealing with the distribution of alkaline phosphatase in different tissues under normal and pathological conditions, including several papers describing its localization in the gingiva.[3,10,11,13,14,15] This was due mainly to the consistent results obtained with the alkaline phosphatase technic.

The original Gomori technic for acid phosphatase,[5] on the other hand, has given variable and inconstant results, of difficult interpretation. Recently, other technics for acid phosphatase have been described, based on the demonstration of the alcoholic part of the phosphate ester used as substrate, by means of its conjugation with azo-dyes which precipitate in the sites of acid phosphatase activity.[2,7,12] The importance of fixation and embedding in the loss of acid phosphatase has recently been assessed, special importance being given to cold formaline solutions and the use of frozen sections to avoid the loss produced by dehydration and embedding.[9]

With these improved technics, results are more reliable, and more definite knowledge can be derived from them.

Reports on the distribution of acid phosphatase in skin describe a relation between keratinization and enzymatic activity.[1,8] An accurate understanding of the process of keratinization and its variations in the gingival tissues is essential in periodontal pathology. The knowledge of the distribution of the different enzymes in the gingival epithelium will help clarify our understanding of the process.

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MATERIAL AND TECHNIC

In ten patients, twelve gingival biopsies were taken from places which appeared clinically normal and areas with varying degrees of inflammation. Gingiva, cheek and tongue tissue from one autopsy case were also studied. The material was fixed in 10% formalin at 4°C and frozen sections were cut within 12 hours of the surgical excision. Sections were incubated in Gomori's solution[6] for periods of 30 minutes, one hour, three hours and twenty-four hours. In each case the specificity was controlled with an incubation without substrate and the enzyme destruction with water vapor at 100°C.

FINDINGS

Acid phosphatase activity was found almost exclusively in the epithelium. After one half hour of incubation the enzymatic activity is seen as a thin band in the surface of parakeratinized epithelium or below the horny layer in keratinized epithelium. Usually the keratin layer is devoid of acid phosphatase. Increasing the incubation time the enzymatic activity extends into the deeper layers. First the enzyme is seen in the nuclei, probably due to diffusion, but later all the cell is stained. With 24 hours of incubation all the epithelium gives an intensely positive reaction (Fig. 1).

The intensity of the reaction is somewhat variable in different cases although the topographical distribution always follows the pattern described above.

In the epithelium that lines the lateral wall of gingival sulci and periodontal pockets and in the epithelial attachment, an absence of acid phosphatase was noted, with the exception of sections incubated for long periods of time in which a moderate reaction was seen in all layers of the epithelium (Figs. 2 and 3).

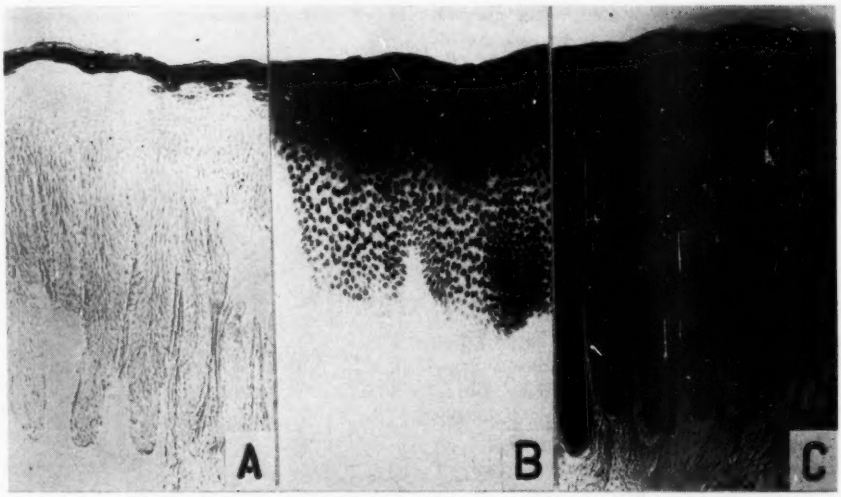

Fig. 1. Histochemical comparison of acid phosphatase activity in gingival tissue after one hour (A), 3 hours (B) and 24 hours (C) of incubation in Gomori's solution. Original magnification × 115.

An epithelial cyst found in the gingival corium of one case had enzymatic activity in its epithelial wall (Fig. 4). The dorsal aspect of the tongue which shows an irregular keratinization due in part to the presence of papilli, shows also an irregular distribution of acid phosphatase which increases in the areas of heavier keratinization. It is also noteworthy the finding of acid phosphatase in the salivary glands of the tongue mucosa.

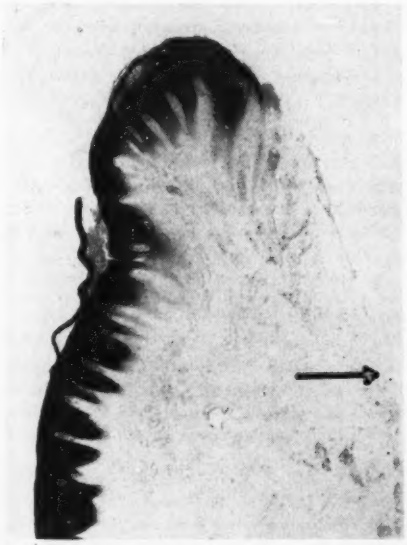

Fig. 2. Low power survey of gingival biopsy after 3 hours of incubation in Gomori's solution. Note the great reduction in activity in the lateral wall of the pocket and in the epithelial attachment. The arrow indicates the deepest portion of the epithelial attachment. Original magnification X37.

DISCUSSION

There is a marked difference between the distribution of acid and alkaline phosphatase in the gingiva.

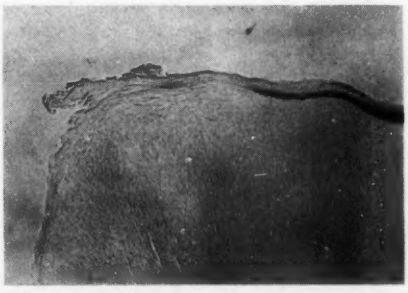

Fig. 3. Gingival margin of biopsy incubated in Gomori's solution for one half hour. Note the thin band of positive reaction situated below the horny layer and ending at the crest of the gingival margin. Original magnification × 115.

Acid phosphatase is seen in the gingival epithelium while alkaline phosphatase is found almost exclusively in the gingival connective tissue. The presence of alkaline phosphatase has also been described in the epithelium, in small amounts, when nucleic acid is used as substrate instead of sodium glycerophosphate;[3] nucleic acid probably lowers the pH during incubation.

There seems to be a relation between acid phosphatase and keratinization. The areas of greatest enzyme activity are the layers immediately subjacent to the horny layer, where biochemical changes leading to keratin formation are expected to be taking place. Once the keratin is formed the enzyme disappears totally or partially. In nonkeratinized areas a great reduction of acid phosphatase activity is seen.

The results obtained in this study are comparable to those described by Bejdl[1] and Moretti and Mescon[8] in their reports on the distribution of acid phosphatase in the skin.

Fig. 4. Epithelial cyst found in gingival biopsy incubated in Gomori's solution for 3 hours. Note the great activity in its epithelial wall. Original magnification × 115.

SUMMARY AND CONCLUSIONS

Histochemical study of acid phosphatase in normal and inflamed human gingiva showed that this enzyme is located almost exclusively in the epithelium. There is a greater reaction in the superficial layers (with the exception of the horny layer which is acid phosphatase-free) and a gradual diminution towards the basal cell layer. The reaction is absent or very weak in the epithelial lining of the gingival sulci and periodontal pockets and in the epithelial attachment.

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Considerations Governing the Choice of Treatment of Periodontal Diseases

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THE list of techniques used in the treatment of periodontal diseases is a long one and includes such diverse measures as scaling; subgingival curettage of varying techniques; complete occlusal equilibration; spot grinding; nutritional fortification; various techniques for performing periodontal surgery; the use of chemotherapeutic and antibiotic aids, caustics, solvents, oxygenating substances, surgical packs; ligation and splinting; and a great variety of adjunctive measures for home care.[2,6] In addition, from a study of the many entities included under the category "periodontal diseases," based upon the various causal and pathologic considerations, it becomes evident that treatment of any one specific entity of this large group of diseases cannot include all of the previously mentioned procedures. It is also evident that one therapeutic procedure cannot be the panacea for all varieties of these diseases. Rather, proper management must be based upon careful evaluation of all subjective and objective considerations indigenous to each patient and tailored to those specific needs in order to achieve the optimum in patient health, comfort, and esthetics. It is the aim of this paper to explore the various factors which play a part in the formation of the ultimate treatment plan.

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CONSIDERATIONS GOVERNING CHOICE OF TREATMENT

Conscious Considerations:

1. Fulfillment of objectives of periodontal therapy. (Miller[11])
   a. Elimination of the causal factors.
   b. Elimination of the effects of causal factors upon the periodontium.
   c. Maintenance of the health of the periodontal tissues at their maximum level.

2. Tissue response to irritants.
   a. Inflammation with incomplete resolution.
   b. Inflammation resulting in fibrosis.
   c. Combination of above responses.
   d. So-called degenerative periodontal disease, as seen in precocious advanced alveolar atrophy and periodontosis.

3. Subjective considerations.
   a. Periodontal health.
   b. General health.
   c. Comfort.
      (1) Minimal preoperative anticipation.
      (2) Minimal pain and discomfort during treatment.

(3) Minimal pain and discomfort following the completion of treatment.

d. Esthetics.

Conditioning Effects upon Choice of Treatment

1. Background of therapist.
   a. Skill, knowledge, education and experience.
   b. Mental and physical health.
   c. Motivation.

SELECTION OF PERIODONTAL THERAPY

The therapeutic regimen for each patient should be determined by the selection of procedures which will best satisfy the three main considerations as outlined above: fulfillment of the objectives of periodontal therapy; observation of tissue response to irritants; and compliance with the subjective considerations of the patient. To accomplish this, it is suggested that the treatment of choice be selected for each section of each category in the above outline. Thus at the conclusion of the pretreatment analysis, a combination of procedures or total composite treatment plan will be evolved. Should a conflict arise between the component selections, for example, between the choices of subgingival curettage and gingivectomy, a study of the complete plan with all considerations in mind will help to resolve the problem and determine the order and selection of procedures.

FULFILLMENT OF OBJECTIVES OF PERIODONTAL THERAPY

The objectives of periodontal therapy are the elimination of the causes and effects of periodontal pathology, and the post-treatment maintenance of the supporting tissues in order to prevent future ravages and destruction similar to those noted pre-operatively. Every case of periodontal disease is the result of the interplay of multiple causes, rather than the effect of a single causative factor.[11] Miller has categorized these causes which are universally present in varying combinations into three groups: dysfunctional, irritational, and systemic.[11] Irritational factors comprise hard and soft deposits on the teeth primarily and the improper use of exogenous agents such as the toothbrush, toothpick, and dental floss. Dysfunctional factors are concerned with either excessive or insufficient occlusal and incisal stresses during masticatory and non-masticatory mandibular movements. Included in the non-masticatory patterns are tensional clenching, swallowing, and various occupational and neurotic habit patterns.[17]

In every case of periodontal disease the systemic component plays either a favorable or unfavorable modifying role.[3] Thus, favorable constitutional integrity of epithelium, and the mesodermal derivatives including connective tissue, bone, and cementum will greatly limit the progress of periodontal disease in the presence of local irritants. Systemic factors which may modify the periodontium unfavorably are gonadal factors as seen during adolescence, pregnancy, the menses, and the menopause; diabetes; thyroid disturbances; parathyroid disease; and vitamin deficiencies, to mention but a few.

Therefore, because all three causative groups, dysfunctional, irritational, and systemic, are present in varying degrees in all periodontal diseases, the treatment of this phase consists of the elimination of these respective causes.[11] The deposits and other types of debris are removed by thorough scaling and subgingival curettage, and the dysfunctional factors are corrected by carrying out the steps of occlusal equilibration consistent with the requirements and age of the patient. If an unfavorable systemic component is present it is improved to as great a degree as is possible by necessary medical care.

The local results of periodontal pathology are usually pocket formation; thickened fibrotic margins and other types of gingival enlargements;[3] gingival cleft formation; gingival recession; alveolar bone changes; tooth mobility; and malpositioning of teeth. Much of the periodontal liter-

ature has been focused on therapy for the elimination of these various results of periodontal disease, primarily on techniques for pocket elimination. Recently, the mechanisms conducive to the malpositioning of teeth and their subsequent reactive positioning have been elaborated upon.[14] Reactive positioning refers to the inherent tendency of malposed teeth to assume a more favorable position in the dental arch with the elimination of those blocking and moving forces which originally contributed to the malpositioning. It is recognized that all teeth undergo some degree of vertical and/or horizontal movement during contact with their antagonists, and that these teeth usually return to their original positions following the breaking of this contact. However, following the soft tissue changes of periodontal diseases and the repeated traumatic insults of antagonists, some teeth move away from their original positions. Following the elimination of the dysfunctional and irritational causal factors and their resultant tissue changes, many teeth once again assume more favorable positions. Reactive positioning is endogenous in character and occurs without the introduction of any exogenous orthodontic aids.

The final aspect of the "objectives" to be achieved is the maintenance of the health of the periodontal structures at a point where they will be better able to withstand future tissue insults. This is accomplished by various means of home care of the gingiva and teeth as the Stillman and Charter methods of toothbrushing; the use of the rubber-tip and other stimulators; and the use of the irrigating syringe. Periodic maintenance, including scaling, sub-gingival curettage, occlusal re-evaluation and correction, re-examination of home care techniques, and careful tooth polishing is a most important consideration.

TISSUE RESPONSE TO DYSFUNCTIONAL AND ENVIRONMENTAL IRRITANTS

In preparing the treatment plan for the patient, the next factor to consider is the type of basic tissue reaction resulting from the irritants. This reaction is usually inflammatory in nature and varies in its end result depending upon the degree of healing and fibrotic scarring which has occurred. Tissue responses may vary from massive chronic inflammatory changes with complete destruction of the epithelium and connective tissue corium, through the formation of incompletely healed highly vascularized granulation tissue, to the production of generalized fibrous connective tissue formation with a minimum of residual inflammation. While this last result may frequently appear to represent the most favorable response, this is not always the case as clinically such tissue may be markedly thickened, may act to entrap food, and may in itself become a causal factor of periodontal disease.[5] The final type of tissue change is the so-called degenerative type seen in precocious advanced alveolar atrophy and periodontosis, where local factors are minimal during the early stages of the disease and where some inherent predisposing factor seems to have overwhelmed the alveolar and soft tissue supporting structures, rendering them so defenseless as to be unable to withstand even moderate irritation.[15]

Generally, when considered in relationship to the tissue response phase, treatment should be directed toward the transformation of the labile states into more stable states by utilizing techniques which involve minimal tissue removal. Thus, areas which show overwhelming inflammatory change, a labile state, should be treated by scaling and curettage rather than by periodontal surgery in order to reduce the changes associated with inflammation and its by-products. The gingiva will then be converted to a more resistent state characterized by repair in the gingival corium and cervicular epithelium. When areas characterized by thickened fibrotic margins and by pocket formation with minimal clinical evidence of inflammation are present, gingivectomy and gingivoplasty should be ac-

complished after the removal of the causal agents previously discussed.[4,5] However, a thickened margin may be retained if it is esthetic, does not induce food impaction, and does not form the outer boundary of a crater. In such a case, the gingival festooning may be a favorable compensatory response to previous irritational factors.

Therapy in the precocious advanced alveolar atrophy patients should include the extraction of the hopelessly involved teeth; the reduction of the superimposed inflammatory reaction; the removal of all local irritants to as great a degree as possible; the institution of appropriate systemic therapy; and finally the stabilization of the remaining healthy teeth with appropriate splinting procedures.[12]

SUBJECTIVE CONSIDERATIONS

A tremendous amount of thought has been expended in the planning and execution of procedures designed to satisfy the objectives of periodontal therapy as outlined previously. Unfortunately, however, the patient's needs have not always been so exhaustively considered, and as a result, a satisfactory and acceptable clinical result has sometimes proven to be less than satisfactory to the patient. Therefore, the important subjective considerations of optimum health, optimum comfort, and optimum esthetics must be planned for prior to the initiation of therapy.

PERIODONTAL AND GENERAL HEALTH CONSIDERATIONS

Optimum periodontal health needs no additional amplification in this phase of the discussion, because it is felt that a major part of the material up to this point has dealt with those considerations which are directed toward achieving optimum health. This is probably the most frequent and most important reason for patients to seek periodontal therapy and it is also the prime objective of the therapist.

In addition, the general status of the patient must be carefully considered in preparing the ultimate treatment regimen. Such factors as physiologic and chronologic age, general health, and emotional stability will all contribute to the type of response to therapy. Periodontal diseases involving alveolar bone loss in a young individual usually progress far more rapidly than do similar conditions in the adult and suggest a more unfavorable prognosis for the involved teeth than would a similar amount of bone change in an adult.[3] Therefore, if the bone loss has progressed to a vulnerable area such as a molar bifurcation or trifurcation, extraction would be the indicated treatment for such teeth, with subsequent replacement in order to protect the adjacent teeth.[11]

While gingival disease not involving the underlying supporting bone is characterized by a more marked inflammatory reaction in children than in adults generally, it is recognized that these areas frequently resolve adequately with treatment and the prognosis here is favorable. Recurrences are likely, but these become less frequent as the patient enters adult life. At the opposite end of the scale, true senility would tend to militate against extensive periodontal care. Aside from these age considerations, the great majority of patients from childhood to late adult life would be eligible for routine periodontal care. As for other limiting factors, specific systemic diseases as discussed earlier will modify the type of treatment consistent with the disease. In addition, even moribund patients such as those with chronic leukemia who seek periodontal care, should receive this type of therapy in order to maintain their oral health and their morale at its highest potential.

The emotional status must also be carefully considered, for if a patient is extremely disturbed extensive periodontal care may heighten this disturbance. Palliative periodontal measures should be used until the patient is emotionally receptive and better able to undergo complete treatment. In this way, every effort will have been made to prevent the additional mental

and physical burden of the loss of the teeth.

COMFORT

While the importance of fear and anxiety to an individual is self-evident, it would seem that the prevention of these emotional states has frequently received far less attention than their control in treating periodontal disease. The benefits of sedatives and relaxants are many and these drugs should be used judiciously, when indicated. However, it would seem logical to avoid their use when possible because of potential untoward physiologic and psychologic effects, if treatment procedures can be selected which produce beneficial results in the periodontium and which at the same time minimize the disadvantage of emotional upset.

Fear and anxiety may be manifested at several stages during the patient-therapist relationship; namely, prior to the diagnostic visits because of anticipation of a potentially unpleasant experience, and prior to and during treatment visits. During the diagnostic phase, the dentist is able to minimize potential emotional trauma by his positive and optimistic approach, emphasizing the brighter side of the evaluation and of the potential result regardless of whether that result implies retention of all, some, or none of the teeth. For example, even if so extreme a recommendation is indicated as the extraction of all remaining teeth, the benefits of complete elimination of the periodontal disease which has reached a terminal stage may be highlighted, and thus permit the patient to accept the best possible solution far more hopefully.

During the treatment phase, these emotions are again minimized by the therapist's optimism, self-confidence, and empathy, and his selection of procedures which most efficiently and least painfully accomplish his objectives.

Pain may be experienced both during and following treatment and may persist, particularly in patients with an extremely low threshold of pain, for hours, weeks, or even months.[13] Inasmuch as the implications of surgery in any part of the body frequently produce fear, it is important for the periodontist to plan his treatment in such a way as to minimize the surgical phase if at all feasible. Of course it is not always possible to accomplish this and to still realize the objectives of periodontal therapy as discussed previously. However, when it is remembered that almost all periodontal diseases are inflammatory in nature and represent changes in contour, consistency, texture, and position of the constituents of the periodontium as the response to the dysfunctional, irritational, and systemic causes, it may then be seen that the elimination or correction of these causes and the restoration of physiologic function will markedly improve the anatomic defects. Further, when it is remembered that periodontal diseases are not neoplastic in nature and that the immediate surgical elimination of various parts of the periodontium is not necessary to save either the life or the teeth of the patient, it again seems reasonable to adopt the least traumatogenic treatment methods available. As tissue response is frequently unpredictable, reasonable opportunity should be given for diseased tissue to undergo repair and regeneration. After the inflammatory state has been reduced to its minimum, the tissue should be re-evaluated to determine whether the remaining architecture is physiologically acceptable or whether it will in turn become a cause for future disease. If physiologic gingival architecture is not the result of the previous phases of treatment, this tissue should then be reshaped to establish a harmonious and resistant form.[4,5]

ESTHETICS

Esthetics is the fourth consideration with which both the therapist and patient are deeply concerned, and although it has been placed last in this group, it is not to be considered as the least important. The ultimate appearance of both the teeth and the gingiva must be carefully considered as the postoperative changes may produce

either a harmonious or disharmonious result. Judicious reshaping of anterior teeth can reduce or even eliminate abnormalities such as over-lapping of contiguous teeth, rotation, extrusion, and deep over-bite relationships depending upon the degree to which the malpositioning exists.[11,14] In addition, unesthetic defects in the shape of the individual tooth, such as those due to incisal chips and abrasions, can be eliminated. As gingival position and contour may also contribute to the appearance of the patient, post-treatment regression of massively inflamed and enlarged gingiva may greatly enhance esthetics. On the other hand, additional post-treatment recession of labial gingiva which has already undergone previous recession or pocket formation may sometimes detract from rather than enhance appearance. This last point demonstrates very well the necessity for thorough pretreatment planning and patient education to eliminate potential conflict between the end result as visualized and desired by the patient and the successful outcome based on elimination of the periodontal disease as anticipated by the therapist. As an example, if anterior maxillary or mandibular labial and interproximal pockets exist with minimal inflammation the decision must be made as to whether it is desirable to reduce the pockets to zero depth surgically, thereby exposing a considerable portion of the root surface; or whether it is more desirable to allow some pocket depth to remain as a concession to esthetics.

The interesting recent animal experiments of Waerhaug,[18] offer the conclusion that a soft tissue cuff always forms coronal to the point of tissue-tooth attachment and bear out the concept that it is permissible, even desirable, to allow some sulcus depth to remain.[8,9] Clinical observation on humans has demonstrated that shallow pockets in accessible areas that are subjected to the massaging effects of mastication and toothbrushing may be maintained over a long period of time without apparent increased periodontal destruction.[7] These findings lend evidence to the belief that shallow pocket areas around labial surfaces of the anterior maxillary and mandibular teeth are usually more desirable than excessively denuded and unsightly anterior teeth with shallower gingival crevices.[10,12]

CONDITIONING FACTORS

Conscious planning of periodontal therapy, or any other form of therapy in dentistry or medicine is, of course, influenced by several basic conditioning factors specific to each therapist. It is not within the scope of this discussion to delve deeply into the psychological motivations which militate for specific types of activity on the part of the individual. However, it must be recognized that even the most objective decision in any phase of human life must be modified, subconsciously, by background conditioning factors.

"Our minds are powered by (1) the biologic drives and processes of our bodies and also (2) by the conditioning influences of our childhood and (3) by the circumstances of life to which we must adapt."[1] The therapist, in this case the dentist, has two major areas which color his professional opinion, his educational and professional experiences and his non-professional experiences of childhood and adult life. His professional conditioning is dependent upon the degree and type of emphasis placed on periodontia in the undergraduate curriculum of his dental school, and upon the post-graduate training to which he has been exposed. In view of the great diversity of therapeutic techniques utilized in this field and the variations of enthusiasm by various clinicians for specific techniques, it would seem plausible to assume that his thinking would be markedly influenced by both the knowledge and personality of his teachers. Next, his clinical experience with the techniques will determine to what extent he will utilize them in the future. A satisfactory result with a specific technique will encourage its use, while an unsatisfactory result may eliminate this method from his armamentarium, despite the fact that this fail-

ure may very possibly result from the improper or incomplete use of the technique or its use for a condition in which it is not indicated. Finally, although the result might be satisfactory clinically, unfavorable reaction by a specific patient may prejudice the dentist against the technique for future use.

Non-professional influences on the dentist are the same as those which may affect any other individual. The fact that a person practices periodontics does not, of course, immunize him against the various mental and physical stresses of living. To quote from Saul, "Exploring personalities further, it was quickly found that no one is free of inner tensions; no one is free from conflicts; no one is on perfect terms with himself, with his conscience, with his parents, brothers and sisters, with his wife and children, with his friends and associates. The elements of neuroses, emotional tension and emotional conflicts are in everyone, and these inner tensions and conflicts come out in innumerable forms. . . ."[15] Therefore, it is apparent that conditioning factors may markedly alter clinical judgment.

The prime non-professional conditioning factors are the past and present general health, emotional status, age, and motivating forces. Specifically, if the therapist has an illness which does not allow for the same freedom of movement and digital skill which he previously enjoyed, his choice of treatment may be modified. Again, in another individual, if emotional disturbances are present to a marked degree whether in the professional or non-professional phases of his life, he may suggest a different program than if he were consulted during a calmer period. Age may play its part in the pattern of treatment selection because of the individual's changing philosophy with his increase in years. The younger clinician frequently searches, experiments, and adopts while the older practitioner may resort to a more rigid routine which has evolved from long experience and which has proven itself reasonably successful. Finally, the motivations of an individual may either consciously or subconsciously affect the type of treatment. Various drives such as the desire to heal; the desire for recognition, acceptance, and gratitude in the professional as well as the non-professional community; and the desire for financial security all may play a part in the procedures selected for the treatment of a patient with a periodontal disease.

SUMMARY

The two primary fields of consideration which govern the choice of treatment of periodontal diseases are as follows:

Conscious Considerations

1. Fulfillment of the objectives of periodontal therapy.
2. Selection of treatment in accordance with the type of tissue response of the patient.
3. Compliance with the patient's needs and desires, relative to optimum health, comfort, and esthetics.

Conditioning Factors—Background of Therapist

1. Skill, knowledge, education and experience.
2. Mental and physical health.
3. Motivation.

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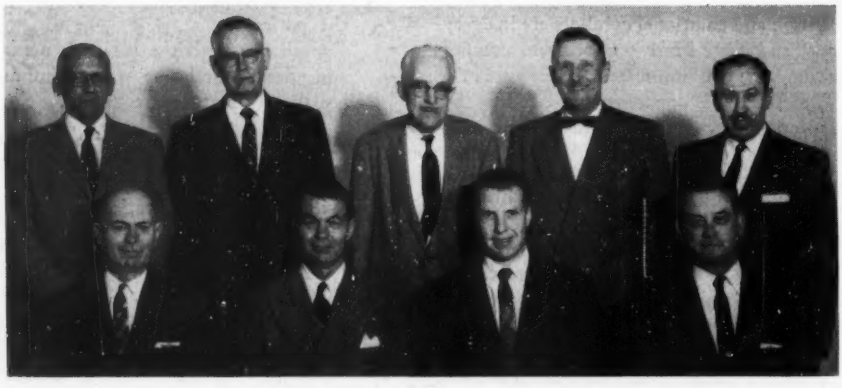

1958 OFFICERS OF THE CALIFORNIA ACADEMY OF PERIODONTOLOGY

The 1958 officers of the California Academy of Periodontology are as follows:

1st row, reading from left to right—Robert L. Rosenberry, President; James L. Pfister, Second Vice President; Robert E. Oslar, Third Vice President; Clarence E. Butler, Treasurer. Jean P. Cantou, First Vice president (not in picture).

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Information regarding this Academy, which is the United States Section of ARPA Internationale, may be obtained by writing the Secretary, Dr. Elwood Wait, 450 Sutter Street, San Francisco, California.

Clinical Uses of Ultrasonic Root Scalers

by Sol J. Ewen,* d.d.s. and Paul J. Tascher,** d.d.s., *New York, N. Y.*

In a recent article the authors[1] describe a method of root scaling based on the use of ultrasonic vibrations. The purpose of this article is to present more recent developments in instrument design and clinical application. The ultrasonic instruments have been used for therapy in dental prophylaxis, marginal gingivitis and the debridement of the hard tissue side of pockets associated with marginal periodontitis.

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**Section Head—Operative Dentistry, Department of Dentistry, The Long Island Jewish Hospital.

Previous investigators[2,3,4,5] have intimated that ultrasonic methods are applicable to periodontal therapy. Richman has described the use of traditional instruments such as: Morse scalers, Zirfing chisel scalers, McCall's No. 1 chisel and Gracey finishing curettes, etc., which were brazed to the threaded hub of an ultrasonic instrument.

The tool tips originally described by the authors[1] in no way resembled the traditional periodontal scaler or curet. These new tips were able to enter easily between the gingiva and the tooth, were blunt to avoid injuring cementum, were contoured so as

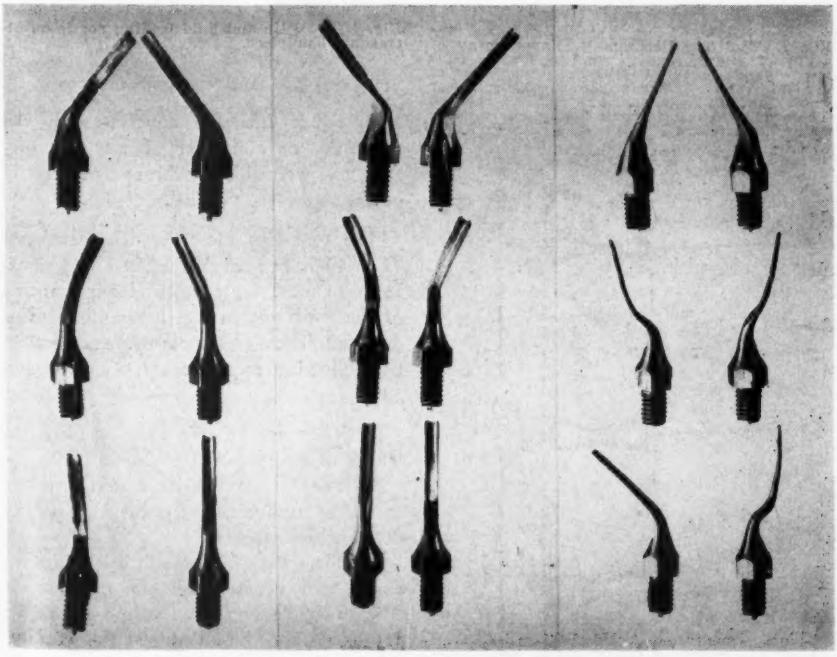

Fig. 1. Ultrasonic Tool Tips
Upper row. Lower posterior right and left ultrasonic scalers shown from various angles.
Middle row. Upper posterior right and left ultrasonic scalers shown from various angles.
Lower row. Lower universal anterior ultrasonic scaler—on left in each column.
Upper universal anterior ultrasonic scaler—on right in each column.

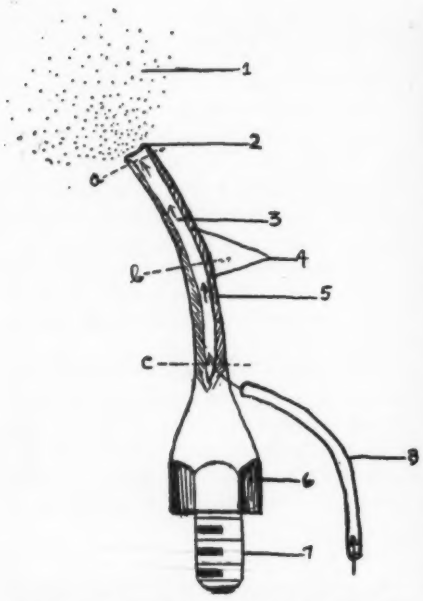

Fig. 2. Tool Tip Diagram—1. Vapor spray of water. 2. Blunt, concave working tip. 3. Hollow ground center. 4. Millimeter markings (2 mm intervals). 5. Arrow showing direction of water flow. 6. Hexagonal hub end. 7. Threads for attachment to ultrasonic handpiece. 8. Nozzle for fluid feeding.

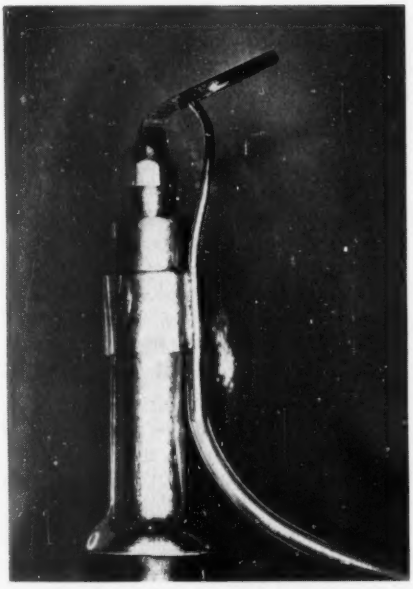

Fig. 4. Tool tip and fluid feeding nozzle on ultrasonic handpiece.

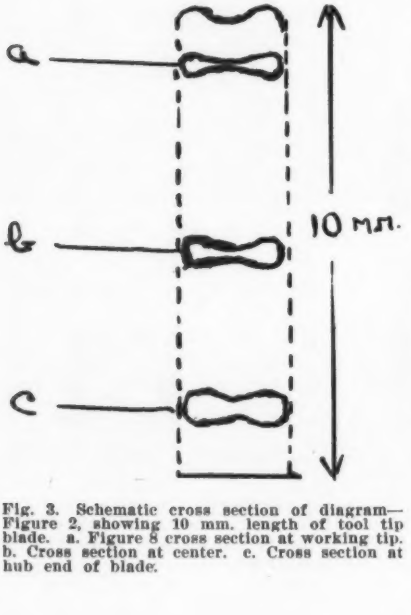

Fig. 3. Schematic cross section of diagram—Figure 2, showing 10 mm. length of tool tip blade. a. Figure 8 cross section at working tip. b. Cross section at center. c. Cross section at hub end of blade.

to produce intimate contact with the convexity or concavity of the root surface, and were able to vibrate satisfactorily at angles off the vertical.

The tool tips most recently developed (Fig. 1) have been devised in a functional form to meet the structural requirements of the tooth within the surrounding periodontium. These ultrasonic root scalers meet the following requirements: (Figs. 2 and 3)

1. They are 10 mm long, exactly the length of a periodontal probe.
2. They are marked at 2 mm intervals to aid in transposing the original pocket depth measurements.
3. They vary in shape in order to have comfortable access to all root surfaces.
4. They taper from the hub end to an approximately ½ mm edge at the working tip.
5. They are hollow ground and in cross section form a figure 8.
6. The blunt working tip is concave.

Fig. 5. Automatic fluid feeding device assembly on ultrasonic dental unit.

When a fluid is continuously fed to a vibrating tip with this design, it is possible to remove surface stains, supragingival and subgingival calculus (Fig. 4). An automatic fluid delivery device propels water and/or antiseptic solutions necessary for the dislodgement of accretions (Fig. 5).

(At this time a further suggestion is undergoing clinical trial (Fig. 6). Results with this new tip will be reported at a later date:

Orthodontic steel tubing of .022 I.D. and .035 O.D. is brazed to an ultrasonic threaded hub. The working tip of the tube is flattened to a Figure 8 cross-section and fluid is forced through the lumen to the working tip. When the ultrasonic power generator is operated the fluid is vaporized at the tip into a fine spray which, upon contact with root surfaces, removes debris.)

CLINICAL PROCEDURES

1. *Prophylaxis:*

With the ultrasonic root scalers in vibration, tap water is directed through a nozzle to the working tip. The patient is rotated in the chair in order to achieve direct visibility of the operative area at all times. It is helpful to use a power evacuator to remove the fluid, although the standard saliva ejector may be adequate. With the use of light rhythmic push strokes the working tip is contacted to stains and supragingival calculus; accumulations are easily dislodged (Fig. 7).

2. *Marginal Gingivitis:*

In treating marginal gingivitis with the ultrasonic root scalers antiseptic solutions are utilized. With automatic fluid feeding, an hypertonic saline solution, or a dilute aqueous solution of buffered sodium hypochlorite (zonite), is force fed to the working tip. With gentle push strokes, placing the instrument directly between the edematous gingiva and root surface materia alba, calculus, plaques and exudate are eliminated. Within 24 to 48 hours (Fig. 8) the gingival papillae are retracted and dip back into the embrasures; color, form and texture reflect signs of healing.

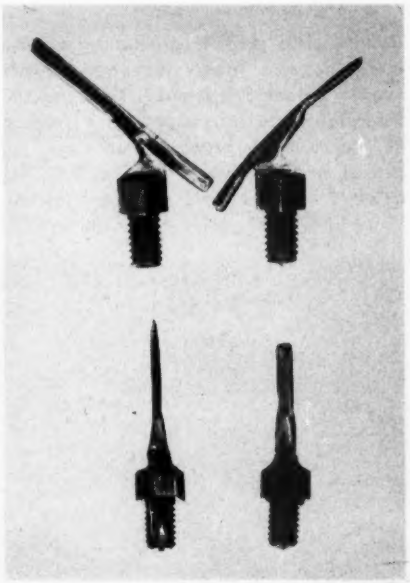

Fig. 6. Tubular ultrasonic tool tip.

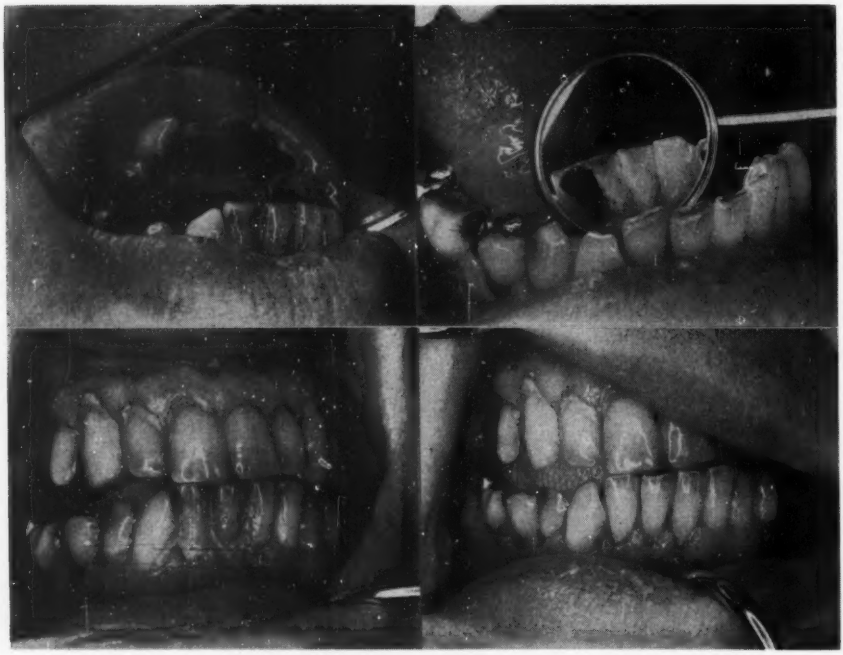

Fig. 7. Left: Prophylaxis—Preoperative views. Right: After prophylaxis.

3. *Marginal Periodontitis:*

With either hollow ground or tubular ultrasonic root scalers each pocket previously measured is entered. Utilizing the marking on the instruments as a measure of pocket depth antiseptics or water are carried to the depth of the pocket. The figure 8 cross sectional shape and widening taper of the instrument retracts the crevicular wall.

The concave working tip is moved in a repeated rhythm of short strokes with continuous overlapping. The concave form of the tip imparts to the instrument the sensitivity and tactile sense of a powered hand instrument.

Fig. 8. Left: Marginal Gingivitis—Preoperative. Right: Post-operative (48 hours).

Histologic studies are being carried out by the authors to establish the adequacy of ultrasonic root debridement in advanced cases of periodontitis.

SUMMARY

A set of ultrasonic root scalers has been described. A method of carrying out dental prophylaxis, treatment of marginal gingivitis and the removal of subgingival calculus has been presented.

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ARPA INTERNATIONALE

The ARPA Internationale convention will be held in Paris at the New Medical Faculty Building, July 7 to 12, 1958. This convention will be of exceptional importance because it will be the 25th anniversary of ARPA Internationale.

Authors who have papers to be presented are requested to address them to Professeur A. J. Held, President de l'Arpa Internationale, Institut Universitare de Medecine dentaire, 24 rue Micheli-du-Crest, Geneva, Switzerland.

There will be numerous festivities besides the scientific meetings, also a Ladies' program.

THE REGISTRY OF ORAL PATHOLOGY

The Armed Forces Institute of Pathology and the Registry of Oral Pathology of the American Dental Association announce an annual slide seminar in advanced Oral Pathology to be held at the Armed Forces Institute of Pathology in Washington, D. C., the first seminar being held on Friday, May 9, 1958 from 9 a.m. until 5 p.m. in Room 351, Armed Forces Institute of Pathology.

This seminar is designed for teachers of Oral Pathology, Oral Medicine and Oral Diagnosis and will consist of a detailed presentation and analysis of diseases and tumors of the oral regions. A set of twenty-five microscopic slides, covering the entities under discussion will be used at the seminar and will then become the property of the seminarian. A descriptive brochure giving history and discussion will accompany the slide set.

For further information write Joseph L. Bernier, Colonel, DC, USA, Registrar and Pathologist, The Registry of Oral Pathology, Armed Forces Institute of Pathology, Washington 25, D. C.

Gingival Pedicle Flaps for Covering Root Surfaces Denuded by Chronic Destructive Periodontal Disease—A Clinical Experiment*

by BENJAMIN PATUR, D.D.S.,** AND IRVING GLICKMAN, B.S., D.M.D.,*** *Boston, Mass.*

DENUDATION of the root surfaces of the maxillary anterior teeth by chronic destructive periodontal disease often presents an aesthetic problem. To overcome this problem, a plastic operation in which the denuded root surfaces are covered by a pedicle gingival flap has been suggested.[1] This paper reports a clinical study to explore the effectiveness of this type of operation.

Seven patients, from 41 to 50 years of age, with varying degrees of chronic destructive periodontal disease served as experimental subjects. Gingivectomy to eliminate the periodontal pockets was instituted in each case. This was followed by a plastic operation in which denuded root surfaces of the maxillary anterior teeth were covered by gingival flaps. The operation consisted of the following procedures (Figs. 1 and 2):

A horizontal incision was made approximating the muco-gingival line extending between the right and left canines (Fig. 2C), and a vertical incision was made in the midline separating the right and left segments (Fig. 2C). (The latter was omitted in one case.)

Individual marginal mesiodistal incisions were made to sever the interdental papillae. The two flaps thus created were elevated, including the periosteum, and interpolated rootward so that a portion of the flap was in contact with alveolar bone and the remainder covered previously denuded root surface. The flaps were then sutured at the midline using 000 silk suture material (Fig. 2C). The margin of the flap was joined to the palatal gingiva which remained in situ by individual interproximal sutures. In the one case in which the vertical midline incision was omitted the gingival tissue was drawn down over the roots as a unit.

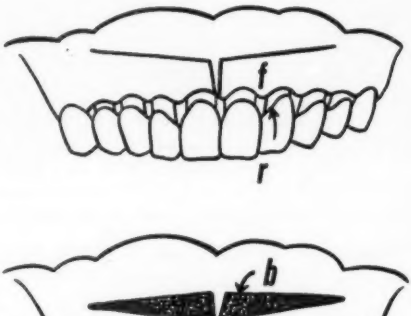

Fig. 1. above, Diagrammatic representation of flap operation showing horizontal incision at mucogingival line and vertical incision in midline. The denuded root surface to be covered is indicated by the arrow (r). The gingival flap is designated by f. below, Flap (f) interpolated onto roots and sutured interproximally and in the midline. Darkened area (b) is denuded bone.

The flaps thus formed were broader at the canine regions in the area of attachment than at the midline, tapering from approximately 6 mm to 4 mm. Immediately above the flaps there was an area of bone which was denuded when the flaps were interpolated downward over the root surfaces (Fig. 2C).

In four patients, the plastic operation was performed immediately following

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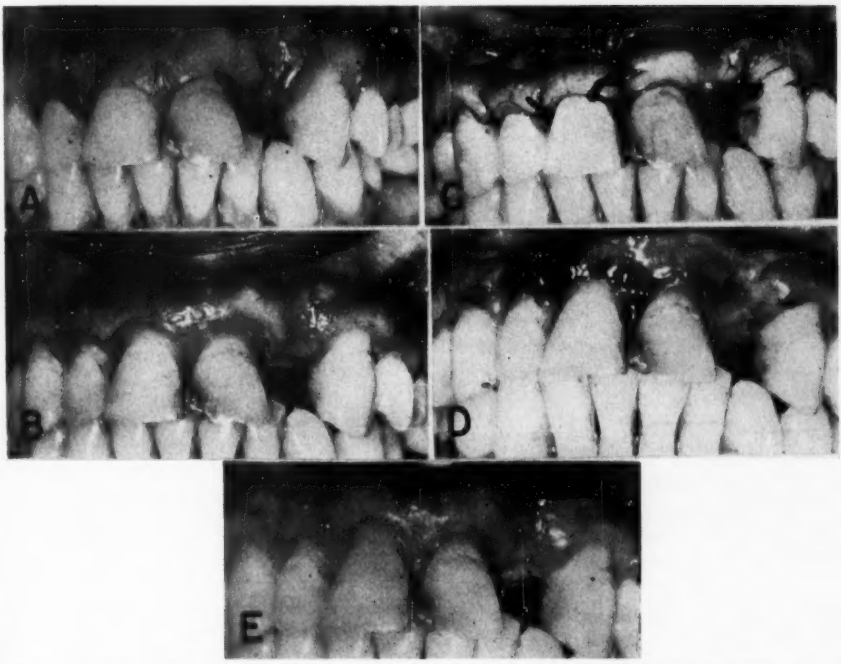

Fig. 2. Various stages in the flap operation. 2A—Anterior maxilla before gingivectomy. 2B—After gingivectomy and immediately before flap operation. 2C—Pedicle flaps interpolated in position on the roots. Note horizontal incision from canine to canine, midline incision and sutures interproximally and at the midline. The bone denuded by the operation is visible above the flap. 2D—Two weeks after flap operation, granulation tissue which replaced a portion of the flap blends with that covering previously denuded area of bone. Note that necrosis and shrinkage of the flap expose root surfaces which were initially covered by the operation (compare with Fig. 2C). 2E—Ten weeks after flap operation. The position of the gingiva on the root surfaces is not appreciably different from what it was before the flap operation was performed (compare with Fig. 2B).

periodontal treatment. In the other three cases, intervals of from five to six weeks were permitted to elapse between periodontal treatment and the plastic operation.

Observations were made at weekly intervals from seven to ten weeks after the plastic operation. These included measurements of the position of the gingiva on the labial surfaces of the teeth in an effort to determine the extent to which the flap operation provided coverage of root surface previously denuded by disease. Using a calibrated periodontal pocket probe measurements were made as follows (Fig. 3):

From the gingival margin to the incisal edge (A); from the gingival margin to the base of the pocket (B). The sum of A and B provided a clinical index of the location of gingival attachment on the individual teeth. Comparisons of the measurements obtained in the course of the experiment indicated whether the level of gingival attachment had been altered.

In the four cases in which the flap operation was performed immediately after the gingivectomy, the measurements were made (1) before any treatment was instituted and (2) at the termination of the experimental period following the flap operation. In the three cases in which there was a time interval (5-6 weeks) between the gingivectomy and the flap operation, measurements were made (1) before gingivectomy (2) after the gingivectomy had healed, immediately preceding the flap operation

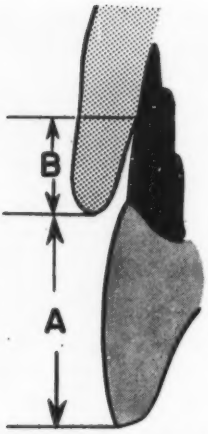

Fig. 3. Diagrammatic representation of measurements made to determine changes in the level of gingival attachment. A. From the gingival margin to the incisal edge. B. From the gingival margin to the base of the pocket. A plus B. Distance from the incisal edge to the position of gingival attachment.

Fig. 4A. One week after operation, showing necrosis of flap.

Fig. 4B. Three weeks after operation, granulation tissue which replaced a portion of flap blends with that covering previously denuded area of bone.

and (3) at the termination of the experimental period.

The flaps underwent degeneration, necrosis and shrinkage so that no appreciable increased root coverage resulted from the operation (Fig. 4A). The portion of the flap which was in contact with underlying alveolar bone appeared to be replaced by granulation tissue, necrotic remnants of the flap becoming surface covering for the granulation tissue and sloughing off between 7 to 14 days after the operation. That portion of the flap which had been placed upon the denuded root was not replaced so that the root became exposed (Figs. 2D and 4B).

The alveolar bone denuded when the flaps were interpolated onto the roots was covered by granulation tissue within the first post operative week. This blended with the granulation tissue which replaced portions of the necrotic flaps as described above. In general, maturation of the granulation tissue to form marginal and attached gingiva occurred between 4 and 8 weeks after the operation. There was a slight linear scar in five of the patients at the site of the initial horizontal incision at the mucogingival junction.

Clinical examination suggested that minor changes had occurred in the position of the gingiva on the labial root surface of several individual teeth. However, our measurements indicated that in general, the flap operation effected no appreciable change in level of gingival attachment (Fig. 2E).

SUMMARY

Subject to the limitations inherent in clinical experiments in humans in which the number of subjects is small, the findings indicate that the pedicle gingival flap operation described here is not an effective method for obtaining coverage of root surfaces denuded by chronic destructive periodontal disease. The gingival flaps did not survive after being sutured in position, and underwent necrosis instead of becoming attached to the root surface.

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Some Alveolar Bone Findings in Hospitalized Patients

by CARL BLACHARSH, B.S., D.D.S.;* DANIEL STAUB, A.B., D.D.S.* AND REUBEN MARGOLIS, B.A.,** *New York City*

PERIODONTAL disease has been accepted as being widespread in our civilization. Stresses have been thought to influence the periodontal tissues via endocrine imbalances,[1,2] metabolic disturbances, tissue destruction by a negative nitrogen balance, etc.[3] Systemic illnesses and emotional illnesses may be considered some of the stressors affecting individuals. In this study we are concerned with the evaluation of these stressors on the alveolar bone.

In the recent past epidemiological studies have been coming to the fore to show the incidence of periodontal disease. Belting[4] in his review of the epidemiology of periodontal disease points out that the peak incidence of disease is reached between 40-50 years of age. He also indicates that roentgenographic examination appears to have the greatest potentiality for providing reproducible or comparable statistical data.

Miller[5] states that "any disease which is capable of reducing the general health of the individual will increase mouth susceptibility to local disorders." There have been many reports as to the influence of generalized diseases upon the health of the periodontium.[6-14] To sum up, the evaluating committee of the periodontal workshop stated that "Systemic influences affect all periodontal disease."[11]

It is believed that emotional factors are etiologically significant in periodontal disease. Various investigations have tied together emotional factors and periodontal involvement.[14-19] It has also been pointed out that there is a positive correlation between the anxiety state and periodontal disease.[20]

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MATERIAL AND METHOD

In a Veterans Hospital in New York all patients who are hospitalized for any length of time and are not immobilized report to the dental clinic where a full mouth series of fourteen roentgenograms is taken. A total number of 169 male patients' roentgenograms were included in this study. (No female patients' roentgenograms were available.)

To conform to the principles of a blind study, the procedure went as follows: On a suitable chart the patient's name and hospital number were recorded. With no knowledge of the patient's age or systemic illness, the investigators (C. B. and D. S.) examined the first molar, cuspid and central incisor in each quadrant of the mouth. This totalled twelve teeth. At least six of these twelve teeth had to be present or the patient was rejected for this study. The roentgenograms of the above mentioned teeth were examined for:

1. The presence or absence of a visible lamina dura at the alveolar crest (both mesial and distal). This was recorded as + or −. From the standpoint of periodontal health the + was desirable.

2. The ratio between the alveolar crest level and the root length starting from the cementoenamel junction.[21] This ratio did not change even if the teeth were elongated or foreshortened in the technique used for roentgenography. This score was recorded for both mesial and distal as 1, ¾, ½, ¼ or 0. 1 was the ideal figure. The numbers represented a ratio only.

3. Slow or rapid alveolar bone resorption (Miller's classification)[22] for both mesial and distal, where present was recorded as plus, or, where there was no resorption, as minus. The latter was the desirable finding where a positive finding denoted the presence of resorption.

All these provided individual tooth scores. To obtain patient scores:

1. The individual lamina dura scores were averaged counting the minuses as against the total number of teeth. This came to a fraction less than one—the closer to one the fraction—the less desirable from the standpoint of periodontal health.

2. The individual alveolar bone scores were averaged for each patient and multiplied by four. This was done to avoid working with fractions. Thus 4.0 denoted a score of perfect health.

3. The slow and rapid types of alveolar resorption were averaged separately. These came to fractions less than one. The closer to one the fraction—the less desirable.

Following determination of the patients' scores the final medical diagnosis and the age of the patient were obtained from the hospital record room.

With the final diagnosis obtained the patients were classified into three age groups, e.g. 20-29 years, 30-39 years, and 40-49 years. Patients over 49 years of age were rejected. The remaining patients were further classified into three experimental groupings:

1. A medical group—consisting of those hospitalized for the more severe and more chronic problems.

2. A neuro-psychiatric group—consisting of those hospitalized on the neuropsychiatric service.

3. A control group—consisting of those hospitalized for relatively non-chronic ailments as fractures, hernias, hemorrhoids, etc.

The distribution of the patients may be seen in Tables 1a and 1b.

The scores were then analyzed statistically with the following variations considered:

1. Age as a factor in affecting the presence or absence of the lamina dura, presence or absence of slow or rapid resorption,

TABLE 1A

Number of Subjects in Each Age Group and in Each Diagnostic Group

	Diagnostic Group			
Age	Medical	Neuro-psychiatric	Control	Total
20-29	16	15	16	47
30-39	34	14	21	69
40-49	26	10	17	53
Total	76	39	54	169

TABLE 1B

Medical Diagnoses of Subjects in Each Age Group and in Each Diagnostic Group

	20-29	30-39	40-49
Medical Group			
Alcoholism	1	2	
Arthritides		4	3
Cardiovascular	1	1	1
Diabetes	3	2	1
Gastrointestinal	7	14	15
Kidney		1	
Liver	2	1	1
Malignancy		3	1
Multiple sclerosis		2	
Tuberculosis	1		1
Other	1	4	3
Total	16	34	26
Neuropsychiatric Group			
Anxiety neurosis	4	1	2
Character disorder		3	
Conversion hysteria		1	
Convulsive disorder		1	1
Depression reaction		1	
Mental deficiency			1
Narcolepsy	1		
Obsession compulsion		1	
Paranoid personality		1	
Paranoid schizophrenia	7	3	4
Psychoneurosis	1	1	1
Schizophrenic reaction	2	1	1
Total	15	14	10
Control Group			
Abscesses and cysts	3	6	
Benign tumors	1	1	
Dislocations & fractures	1	3	4
Hernias & hemorrhoids	1	3	1
Infections like eye infections, tonsillitis, etc.	7	4	4
Surgical			3
Other	3	4	5
Total	16	21	17

and alveolar bone level.

2. The disease groupings for which the patient was hospitalized affecting the level of alveolar bone, the presence or absence of the lamina dura and the presence or absence of slow or rapid resorption.

3. Interaction between age and disease grouping or between 1 and 2.

In analyzing the data we accepted as a significant finding any result where its occurrence by chance was five times per hundred or less.

TABLE 3

*Analysis of Variance of Mean Lamina Dura Scores**

Source	df	Sum of Squares	Mean Square	F	p
Error	160	—	.0024	—	—
Age	2	.022	.011	4.58	<.05
Diagnosis	2	.002	.001	.42	>.05
Interaction	4	.012	.003	1.25	>.05
Total	168	.036	—	—	—

*Walker, H. M. and Lev, J. Statistical Inference. Henry Holt and Co. New York 1953 pg. 381.

FINDINGS

The lamina dura scores for each age group and diagnostic group showed that the visible lamina dura was lost relatively early in life and the average loss varied from 79% to 95%. (See table 2).

TABLE 2

Mean Lamina Dura Scores for Each Subgroup, Age Group and Diagnostic Group

	Diagnostic Group			
Age	Medical	Neuro-psychiatric	Control	Mean
20-29	.803	.853	.797	.818
30-39	.872	.825	.792	.830
40-49	.912	.946	.926	.928
Mean	.862	.875	.838	

An analysis of the mean lamina dura scores demonstrates that differences due to increasing age are significant whereas differences due to the particular disease groupings are not significant. In addition the interaction between the age of the patient and the disease of the patient on the lamina dura of the patient was not significant. (See table 3.)

The mean slow resorption scores for each age group and diagnostic group showed a variation in occurrence of from 68% to 86% of the teeth examined. (There were too few patients demonstrating the rapid type of bone resorption in all age groups and diagnostic groups to be able to compare findings. Therefore, only the slow type of resorption was reported.) (See table 4.)

TABLE 4

Mean Slow Resorption Scores for Each Subgroup, Age Group, and Diagnostic Group

	Diagnostic Group			
Age	Medical	Neuro-psychiatric	Control	Mean
20-29	.675	.737	.722	.711
30-39	.807	.782	.752	.780
40-49	.855	.843	.864	.854
Mean	.779	.787	.779	

The analysis of the mean slow resorption scores shows that differences due to age are significant whereas the differences due to diagnostic groupings and the interaction between age and diagnostic groupings are not significant. (See table 5.)

The mean alveolar bone scores varied from 2.95 to 3.69 where 4.0 was the maximum possible score (meaning all bone present). (See table 6.)

The analysis of the mean alveolar bone score shows that the bone score decreased with age. This decrease in bone scores re-

TABLE 5

Analysis of Variance of Mean Slow Resorption Scores

Source	df	Sum of Squares	Mean Square	F	p
Error	160	—	.0038	—	—
Age	2	.031	.0155	4.08	<.05
Diagnosis	2	.001	.0005	<1	>.05
Interaction	4	.003	.00075	<1	>.05
Total	168	.035	—	—	—

mained the same with increasing age in both the medical and control groups. The neuro-psychiatric group, with increasing age, had more alveolar bone than the other two groups. Thus the interaction effect between age and diagnostic group was significant whereas age alone, or diagnostic group alone was not significant with respect to the alveolar bone score. (See table 7.)

DISCUSSION

In this study all roentgenograms in the dental file were examined. Roentgenograms were rejected where there was less than the required number of teeth present or where the patient was over 49 years of age. Previous alveolar bone studies had indicated that all significant changes had already occurred by the 49 year age level.[4, 10, 23, 24]

The rationale for the use of three teeth in each quadrant of the mouth was as follows:

1. In a previous study—teeth were classified as to their alveoloclastic susceptibilities.[25] The present study method includes teeth from each susceptibility group.

2. These teeth had erupted at various ages.

3. These teeth were subject to most of the influences that could act in the same mouth because of their different locations.

4. These teeth seemed important in determining the prognosis of the entire masticatory apparatus.

TABLE 6

Mean Alveolar Bone Scores for Each Subgroup, Age Group and Diagnostic Group

	Diagnostic Group			
Age	Medical	Neuro-psychiatric	Control	Mean
20-29	3.68	3.56	3.69	3.64
30-39	3.38	3.58	3.51	3.49
40-49	2.99	3.29	2.95	3.08
Mean	3.35	3.48	3.38	

TABLE 7

Analysis of Variance of Mean Alveolar Bone Scores

Source	df	Sum of Squares	Mean Square	F	p
Error	160	—	.0144	—	—
Age	2	.52	.26	.62*	>.05
Diagnosis	2	.03	.015	.04*	>.05
Interaction	4	1.68	.42	29.17	<.001
Total	168	2.23	—	—	—

*Interaction mean square used as error term.

Using these teeth, the results of the findings in this study were that age seemed to be the most important factor in alveolar bone destruction. With increasing age there was increasing destruction. This was borne out by previous studies.[9, 10]

Alveolar bone destruction was not increased by the systemic disease for which the patient was hospitalized—whether this disease was of a somatic or a neuro-psychiatric type. In other words whether the patient was hospitalized for an eye infection or a peptic ulcer there was no change in the alveolar bone picture.

This does not mean that systemic disease is of no importance in the etiology of alveolar bone destruction or that systemic

stressors do not affect the periodontium. It means that all systemic diseases can not be equated insofar as their effects on the alveolar bone are concerned.

An illness may be serious enough to hospitalize a patient, place him under sufficient duress to warrant his being put in a systemic study group, and yet by the criteria used show no demonstrable effect on the alveolar bone. The same, of course, may be said for the neuropsychiatric group. These findings can be explained on the basis of the normal bodily homeostatic mechanisms still being operable. When these mechanisms become inoperable as in advanced malignancies, severe endocrine diseases and various other wasting diseases—the findings might be otherwise. In the patients chosen for this study there was no attempt made to evaluate the severity of their illnesses—whether that happened to be diabetes, peptic ulcer or paranoid schizophrenia.

There are other factors worth noting here which could influence the alveolar bone picture. These patients were receiving treatment for their illnesses in this Veterans Hospital—and if of sufficient duration in other Veterans Hospitals. One of the therapeutic procedures employed in treating the neuro-psychiatric group in this hospital is mandatory oral hygiene—which is enforced by the attendants. This coupled with proper dental care over recurring and long term hospitalization could account for these patients' better alveolar bone picture as compared to the medical and control groups. This points out again the multiplicity of the etiological factors of periodontal disease and therefore of the multiplicity of variables in studying periodontal disease.

It might also be pertinent to point out that in determining the prognosis in periodontal disease—it is not sufficient to say that the patient has a systemic factor and therefore periodontal treatment may not be successful. As with local factors—systemic factors should be evaluated individually for each patient and prognosis determined by the thorough consideration of all influences.

SUMMARY AND CONCLUSIONS

Roentgenograms of 169 male patients in the New York Veterans Administration Hospital were examined in a blind study. These ranged in ages from 20 to 49 years and represented patients who were classified into a medical study group, a neuropsychiatric study group and a control group of hemorrhoids, fractures, etc., which could not be expected to affect the alveolar bone. Mesial and distal surfaces of selected teeth were studied as to the presence or absence of a visible lamina dura and the presence or absence of slow resorption. In addition the ratio of the remaining alveolar crest to the root length was studied. All these scores were averaged for each patient, age group and diagnostic grouping and the findings compared.

It was found that:

1. The medical group had an alveolar bone loss similar to the control group. Both groups showed the same significant bone loss with increasing age.

2. The alveolar bone scores of the neuropsychiatric group did not decrease with age as much as the medical or control group.

3. The visible lamina dura decreased with age and not with diagnostic grouping.

4. The amount of slow resorption increased with age and not with diagnostic grouping.

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Effect of Phenol Camphor on Gingival Tissue

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REMOVAL of the crevicular epithelium is a prerequisite for reattachment of periodontal fibers. For this purpose different means have been advocated: Soft tissue curettage,[12] sodium sulphide,[7,8] phenol camphor,[1,10] and antiformin.[2] A complete removal of the epithelium by mechanical means is obviously difficult to attain even in buccal crevices which are easy of access,[11] and interproximally where the deepest pockets usually are encountered,[6] these difficulties are considerably enhanced. Both clinical[1] and histologic[16] evidence indicate that phenol camphor is a valuable aid in the attainment of reattachment. Sodium sulphide and antiformin have been claimed to act specifically upon the epithelium[2,7,8] but experimental investigations have demonstrated that at least antiformin affects the connective tissue as well.[3,4,5] This, however, does not seem to interfere with the healing.[5]

Reattachment is an established fact but there is still uncertainty as to the best ways and means to attain it. The purpose of the present investigation is to evaluate (1) the efficiency of phenol camphor in removing the crevicular epithelium, alone and in combination with soft tissue curettage and (2) any harmful effect of it.

MATERIAL AND METHOD

Twenty-two teeth in four dogs with healthy gingivae were used for the experiment. Young animals were chosen to make sure that the epithelial cuff ended at the cemento-enamel junction. Thus it would be possible to decide in the histologic sections whether or not any downgrowth of epithelium had taken place as a consequence of the treatment. The animals were anesthetized with nembutal; cotton strings (approximately 2 mm. thick) saturated with phenol camphor (75% phenol and 25% camphor) were pressed into the pockets by means of a thin and flat instrument with a force of approximately 300 grams. Efforts were made to bring the pack to the deepest point of the pocket in the full length of the buccal and approximal surfaces, where it was left for about 4 minutes. In seven pockets in one dog the soft tissue wall was thoroughly curetted subsequent to the removal of the pack, while in the remaining 15 pockets in 3 dogs no curettement was undertaken; in one case the pack was applied around a tooth used for experimental traumatic occlusion. The observation period varied between 2 hours and 39 days; in one case the pack was left in place during sectioning, having been inserted immediately before the animal was killed. The jaws were taken out with great care and immersed in 10% formalin. After decalcification in 5% nitric acid the specimens were embedded in paraffin and sectioned bucco-lingually; the sections were stained, some with hematoxylin-eosin and some with azan.

OBSERVATION

During the first days after treatment the gingival margin appeared red, and moderate exudation from the pockets occurred when pressure was applied. No difference could be detected between the pockets treated with phenol camphor alone and those that got the additional soft tissue curettage. A colorless plaque of varying thickness invariably accumulated on the tooth surfaces above the gingival margin.

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The pack obviously could be pressed down to the cemento-enamel junction, and during this procedure the epithelial lining could be removed or dislodged (Fig. 1). After removal of the pack the soft tissue swung back to its original place, and observations after two hours revealed that the cells of the epithelial cuff, being pycnotic in appearance as the result of the action of the phenol-camphor, adhered to the enamel cuticle (Fig. 2, top right and left). Where the alveolar crest reached above the cemento-enamel junction the pack was not pressed down to the latter; evidence for this was seen in the two hour specimen where the epithelial cells were intact close to the cemento-enamel junction (Fig. 2, bottom left), whereas close to the alveolar crest red blood corpuscles were found between the epithelium and the cuticle, demonstrating that the pack had been pressed down to this point (Fig. 2, bottom right). The fibroblasts of the connective tissue close to the epithelium were thinner than usual, and did not proliferate; neither did emigration of leukocytes take place (Fig. 3, top right). Somewhat further in the connective tissue the endothelial cells and fibroblasts were rich in cytoplasm and between them were erythrocytes and polymorphonuclear leukocytes; the vessels were dilated (Fig. 3, bottom left). Still further in, the pattern of the connective tissue was normal with thick fiber bundles and slender fibroblasts (Fig. 3, bottom right).

After three days the connective tissue close to the enamel did not take the collagen stain in some places although the fiber structure was still visible; there was a sharp line of demarcation towards the collagenous tissue (Fig. 4, bottom right). Inflammatory cells and erythrocytes were present between the fibers.

After 12 days approximately normal connective tissue with distinct fiber structure often lay in contact with the enamel

Fig. 1. Dog 20. Lower left canine. Hematoxylin-eosin. Animal killed immediately after packing. Pocket depth 4.5 mm. Cotton pack reaches to cemento-enamel junction.

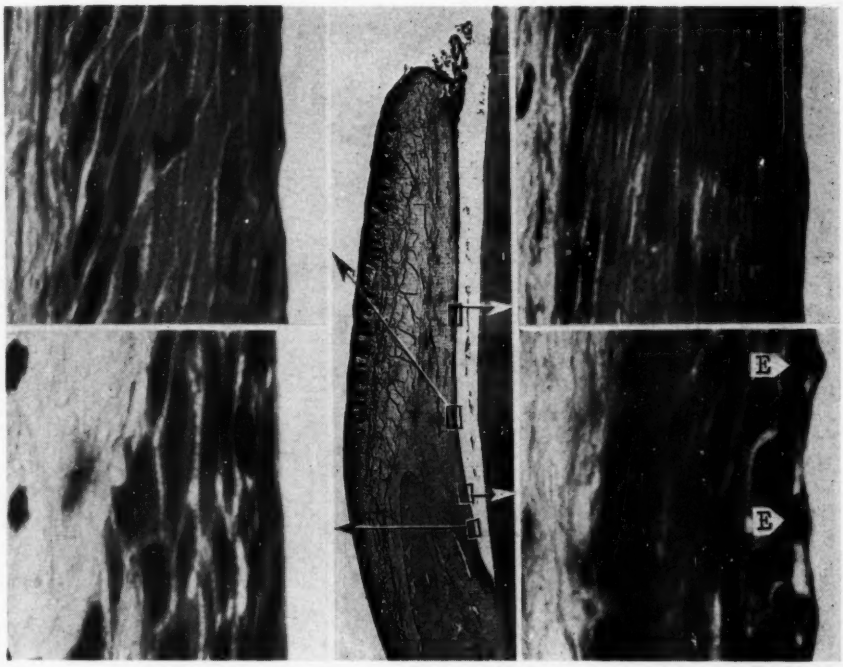

Fig. 2. Dog 20. Upper left canine. Hematoxylin-eosin. Obs. per. 2 hours. Alveolar crest 2 mm. above cemento-enamel junction. Normal epithelial cells adhering to enamel cuticle in deeper part (bottom left). Immediately below alveolar crest are seen erythrocytes (E) between somewhat deranged epithelial cells and cuticle, indicating that epithelium has been reflected (bottom right). Further up epithelial cells are showing pycnotic nuclei in close proximity to enamel cuticle (top, right and left).*

in some places and only few inflammatory cells were present (Fig. 6, bottom right).

Viable epithelial cells were not found close to the cemento-enamel junction after 3 days, but isolated small islands could sometimes be detected in other parts of the pocket (Fig. 5). Proliferation of epithelium from the gingival margin took place regularly (Figs. 4 through 6), and all the pockets where phenol camphor had been used alone, were fully epithelized within 10 days (Figs. 7 through 10). In the beginning some degeneration was present (Fig. 7) but in the later stages the conditions were normal (Fig. 8). In the cases where soft tissue curettage had been carried out, epithelization was apparently delayed as the cervical two thirds of the pockets were uncovered after 10-12 days (Fig. 6). Formation of subgingival bacterial plaque occurred in some cases and corresponding to the plaque the epithelium was degener-

*Although not pertinent to this problem a recent statement by Orban and co-workers[9] should be viewed in the light of Figure 2. They write: "Epithelium is not made adherent to the tooth by formalin fixation." The close adhesion between the epithelium and the enamel cuticle (or between the epithelial cells, if the separation has taken place there) cannot have been caused by anything but the formalin fixation.

In the same article the suspicion was expressed that "by some mistake Waerhaug's sections were made from a tooth or an area of a tooth that had not been subjected to the experimental reflection of the gingival flap." The presence of erythrocytes between the epithelial cuff and the cuticle (Fig. 2 bottom right), and the pycnotic appearance of the epithelial cells caused by the phenol camphor (Fig. 2 top, right and left) as compared with the unaffected cells (Fig. 2 bottom left) are clear evidence that the flap has been reflected; and there is seemingly still an organic union. This shows that the statement of Orban and co-workers is invalid.

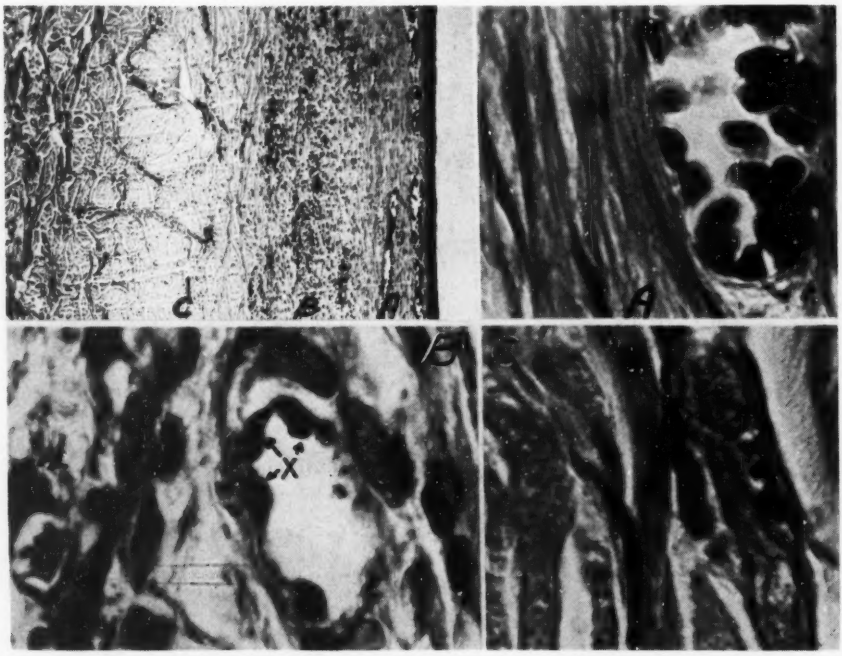

Fig. 3. From area between alveolar crest and gingival margin in same section as Fig. 2. Three layers are discernible: A) Adjacent to epithelium are distended and thrombosed vessels with lack of endothelial lining; fibroblasts are pycnotic and rare; no cell proliferation. B) Adjacent to pycnotic layer fibroblasts and endothelial cells are proliferating; polymorphonuclear leukocytes are lining vascular wall (x), migrating through it (x) or distributed in the connective tissue. C) Outside proliferating layer is unaffected connective tissue without cell degeneration or cell proliferation.

ated and the connective tissue infiltrated with inflammatory cells (Fig. 9). Traumatic occlusion did not interfere with the healing in the pocket (Fig. 10, top right) and the phenol camphor packing apparently did not enhance the effect of the trauma, as the necrosis was limited to the periodontal membrane within the alveolus and just above it (Fig. 10, bottom right); between the alveolar crest and the cemento-enamel junction the periodontal fibers were not affected (Fig. 10, center right).

DISCUSSION

This series demonstrates that cotton strings can be packed to the deepest point of the epithelial cuff when firm force is used (Fig. 1) and this may explain the favorable clinical results with phenol camphor pack. Under favorable conditions the phenol camphor necrotizes the entire epithelial lining of the pocket and the main barrier against reattachment is removed. The drug apparently penetrates the soft tissue and acts as a fixative on the cells (Figs. 2 top and 3, top right). This fixation may explain why fibrous tissue, not taking collagen stain, was present after 3 days (Fig. 4). The phenol camphor imbibition may have retarded the proteolytic decomposition of the dead tissue which under normal conditions should have been absorbed much earlier. However, this retardation apparently does not jeopardize repair, as healing (more or less complete) occurred in all long term experiments (Figs. 7 through 10).

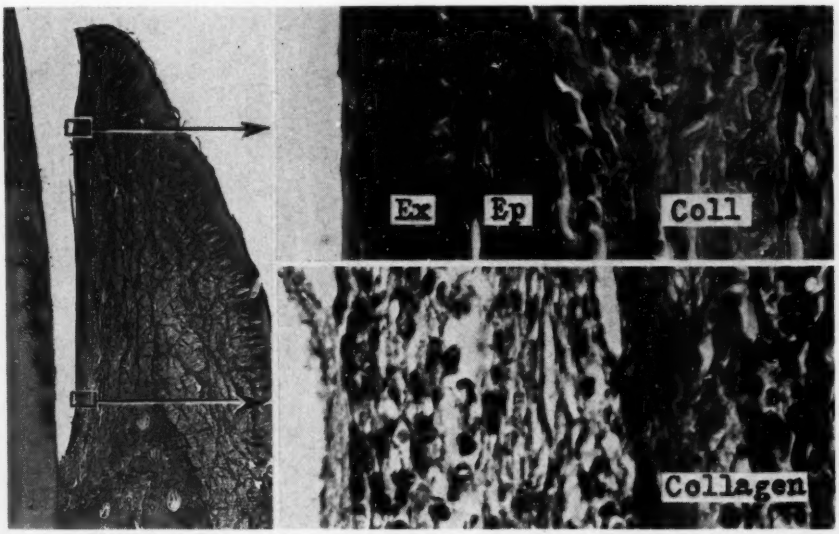

Fig. 4. Dog 19. Lower right canine. Obs. per. 3 days. Azan. Epithelium lacking from slightly below gingival margin (Ep. top right) to cemento-enamel junction which is located somewhat below alveolar crest. Close to remnants of enamel matrix is fibrous tissue not taking collagen stain in close approximation to collagen tissue infiltrated with inflammatory cells and erythrocytes. Exudate (Ex) seen between epithelium and enamel in outer part of pocket (top right).

The effect of the drug apparently is limited by the fibroblasts and endothelial cells forming a barrier of proliferating mesenchymal cells into which migrate polymorphonuclear leukocytes from the dilated vessels (Fig. 3). In this way the necrotic tissue is walled off already after 2 hours (Fig. 2) and repair can start quickly.

The phenol camphor packing alone (Fig. 5) apparently did not remove the epithelial lining of the pocket as efficiently as when combined with soft tissue curettage (Fig. 6), and from earlier experiments it is known that neither is soft tissue curettage effective alone.[11] Therefore, the combination of these two methods may be the choice, and may also explain the good results obtained by Ramfjord.[10] In some parts of the pocket the phenol camphor may be so diluted that it does not necrotize all the epithelium, and the remnants (Fig. 5) will give rise to a quick re-epithelization (Fig. 7); such remnants may, however, be scraped away with the curette.

In no case was there any downgrowth of epithelium below the cemento-enamel junction, even when traumatic occlusion was present (Fig. 10); this shows that serious damage hardly can be expected with this method. However, accumulation of bacterial plaque below the gingival margin obviously was combined with the treatment, indicating that in clinical practice

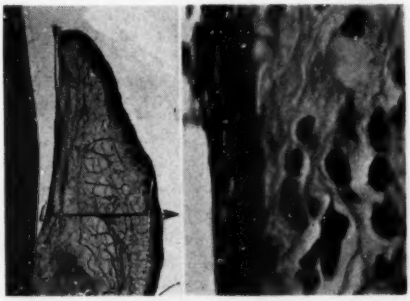

Fig. 5. From same tooth as Fig. 4 but from another part. Hematoxylin-eosin. Epithelium completely necrotized at cemento-enamel junction, but slightly above it are seen viable epithelial cells inside which the connective tissue is infiltrated with polymorphonuclear leukocytes and erythrocytes.

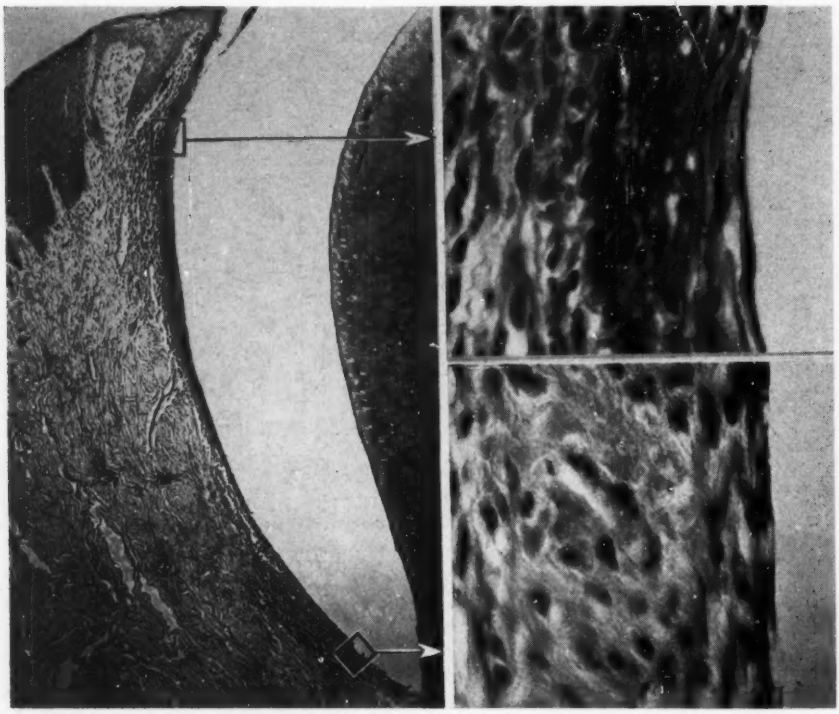

Fig. 6. Dog 5. Obs. per. 12 days. Hematoxylin-eosin. Epithelium absent from cemento-enamel junction (bottom right) to slightly below gingival margin (top right). Connective tissue exhibiting only slight inflammatory reaction adhering to cuticle-like membrane on enamel. Somewhat degenerated epithelium adheres to enamel cuticle as well.

thorough home care should be instituted and cleansing of the teeth in the outer part of the pockets should be undertaken at intervals after the treatment.

There appears to be no basic difference between the final effect of phenol camphor and that of antiformin.[5] Both of them necrotize the epithelium and part of the subjacent connective tissue and in both cases a complete healing may ensue. The main difference may be that the tissue to a certain degree is fixed by the phenol camphor whereas it is liquefied by the alkaline antiformin. Which one is the better cannot be decided until direct comparison has been made.

SUMMARY AND CONCLUSIONS

Twenty-two buccal pockets in four healthy dogs were packed with cotton strings saturated with phenol camphor. In 7 pockets in one dog the soft tissue side was curetted subsequent to the packing. The tissue was allowed to heal for from two

Fig. 7. Dog. 19. Obs. per. 10 days. Epithelization complete, and somewhat degenerated epithelial cells are adhering normally to thickened cuticle.

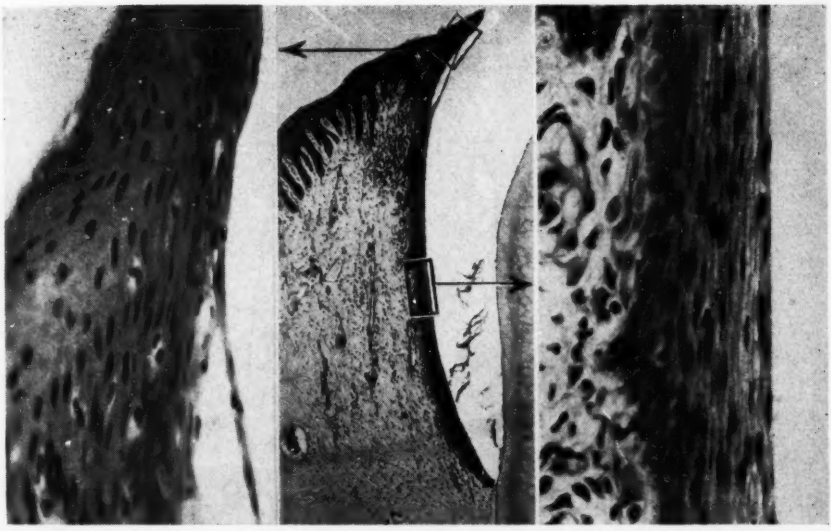

Fig. 8. Dog 21. Obs. per. 32 days. Hematoxylin-eosin. Normal epithelial cells adhere to enamel cuticle from cemento-enamel junction to gingival margin, the split in upper part being an artifact. Inflammatory reaction in connective tissue hardly more pronounced than in control specimens.

hours to 39 days. The following conclusions seem to be justified:

1. A cotton string can be packed to the deepest point of the epithelial cuff if the alveolar crest is not located above it.

2. Phenol camphor necrotizes the epithelium and part of the connective tissue. It acts as a fixative on the soft tissue and may delay proteolysis, but it does not appear to prevent the final healing.

3. The necrotic tissue is walled off by a layer of proliferating fibroblasts and emigrated leukocytes which prevent spreading and start the repair.

4. The epithelial lining of the pocket is apparently more efficiently removed when the phenol camphor packing is combined with soft tissue curettage.

5. Complete healing with re-adaptation of a normal epithelial cuff to the gingival margin will ensue in all cases except those

Fig. 9. Dog 21. Obs. per. 39 days. Hematoxylin-eosin. Epithelization complete. Bacterial plaque formed below gingival margin; adjacent to plaque degeneration and thickening of epithelium as well as pronounced cellular infiltration in connective tissue.

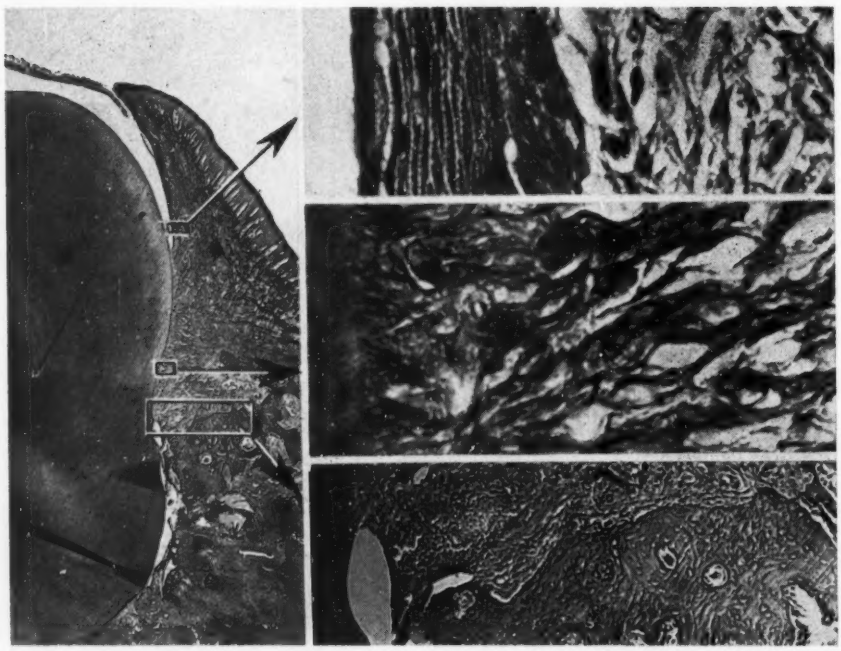

Fig. 10. Dog 21. Obs. per. 25 days. Azan. Tooth exposed to traumatic occlusion during whole observation period. Periodontal membrane and tissue above alveolar crest necrotized (bottom right). Slightly above are seen normal periodontal fibers attached to cementum; no inflammation (center right). Normal epithelial cuff adheres to somewhat thickened cuticle (top right).

where bacterial plaque has been retained on the tooth.

6. Phenol camphor packing facilitates to some degree retention of bacterial plaque for which reason hygienic measures should be taken after the treatment.

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Mast Cells in Gingival Tissue

by MARGARET R. DEWAR, PH.D., *Sydney, Australia*

NORMAL gingival tissue is rich in mast cells and the number of these cells increases significantly when the tissue becomes inflamed.[1] In view of the presence of mast cells in the gingiva and of the interest attaching to these cells, it seems of value to consider briefly the present state of knowledge of this subject.

THEORETICAL

Mast cells are widely distributed throughout the body, especially in organs or tissues rich in connective tissue. Increase in the number of mast cells in chronic inflammation was noted by Erhlich as early as 1879. Considerable local increase in number occurs also in tumors, skin diseases and disturbances of the nervous system.

The function of the mast cell in the body has not been determined, although many functions have been suggested. One of the more recent theories and one supported by Hansen[2] and Sylven[3] is that the mucopolysaccharide of the granules of the mast cells aids in the formation of the intercellular mucopolysaccharide of the ground substance and the chromotrope substance in the epithelium. Investigations of wound healing and pathological tissue rich in mast cells and hyaluronic acid seem to substantiate this theory. However, recent work of Devitt and co-workers[4] shows that changes in the granular content of mast cells observed in fresh and fixed tissue can be caused by methods of fixation and staining. Hansen and Sylven's theory must therefore be carefully re-examined. Cramer and Simpson[5] believe that the increase in mast cells observed in the dermis after application of carcinogenic coal tar derivatives is related to the epidermal hyperplasia. The presence of mast cells in increased numbers in chronically inflamed gingiva may therefore be a sign of healing of the ground substance and the epithelium.

The composition of mast cells is complex. For many years the granules were thought to contain only heparin, a sulphonated ester of a high molecular weight polysaccharide. More recent work has shown the granules to contain also histamine[6] and the cells to contain phospholipid, lipase, acid and alkaline phosphatases, peroxidases and cytochrome oxidase.

The discovery that mast cells contain histamine is of interest when considering the possible mechanism of inflammation in gingival tissue. Histamine, "H substances" and "allergy" have frequently been mentioned as possible explanations of the mechanism and cause of gingival disease,[7] although no experimental evidence has been presented to substantiate these theories. Tissue releases histamine immediately after injury and, when inflammation occurs, the increased histamine content is maintained. Platelets and leucocytes have both been considered vehicles for conveying the additional histamine to the areas of inflammation. The function of histamine is not known but it may initiate the defense response to injury. The fact, that in earlier work the author[1] found histamine-containing cells (the mast cells) in higher concentration in normal gingivae than in most other tissues of the body and in still further increased numbers in inflamed gingivae, is of interest when considering the role of histamine and mast cells in the body.

It has been suggested that one of the first steps in the mechanism of gingival inflammation is the entrance of bacterial hyaluronidase into the tissue.[8] Histamine can increase the spread of hyaluronidase and through this property possibly influence the spread of inflammation. However mast cells contain heparin, a substance which inhibits testicular hyaluronidase 'in vitro.' Whether heparin can inhibit the action of

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TABLE I

Mast Cell Content of Gingival Tissue

Fixative	*Normal Tissue*[1]	*Inflamed Tissue*	
	Number of Mast Cells (per sq.mm.)		
	Connective Tissue	*Connective Tissue*	*Total Tissue*
Lead-acetate-formalin	18.3 (S.D.14.3)	47 (S.D.17)	30 (S.D.11)
Carnoy	—	22 (S.D.11)	14 (S.D. 7)

tissue or bacterial hyaluronidases 'in vivo' is not yet known. But these points are of interest when considering the role of histamine, hyaluronidase and mast cells in tissue and in gingival tissue in particular.

This paper reports the estimation of mast cell and histamine content of inflamed gingival tissue. Normal tissue was unfortunately not available in sufficient quantity for analysis of histamine but comparative figures for mast cell content of normal gingival tissue are available from the author's previous investigation of such normal tissue.[1]

EXPERIMENTAL

Gingival tissue was removed under novocaine anaesthesia from 18 individuals suffering from periodontitis. Small pieces of tissue from each were placed in formalin-basic-lead-acetate fixative and in Carnoy fixative respectively. The remainder was quickly weighed and placed in trichloracetic acid. Paraffin embedded sections (7 μ thickness) were prepared from the fixed tissue and stained by the toluidine blue technique recommended by Kimball.[9] The mast cells present in the *whole* section were counted with the aid of an oil immersion lens, the area was measured and the concentration of mast cells expressed as (1) the number per sq.mm. connective tissue and (2) the number per sq.mm. total tissue (i.e., connective tissue plus epithelium).

The histamine content of the tissue (free and bound) was estimated by the technique of Rosenthal and Tabor[10] and expressed as μ gm. per gm. wet weight of total tissue.

RESULTS

The mean concentration of mast cells in lead-acetate-formalin fixed tissue and in Carnoy fixed tissue is set out in Table I.

Mean concentration of histamine was 60 μ gm. per gm. wet weight total inflamed tissue (S.D. 17). A significant correlation was not obtained between the concentration of mast cells in individual samples and their histamine content, the correlation coefficient being 0.37.

DISCUSSION

These figures show that inflamed gingival tissue is rich in both mast cells and histamine and the results confirm the qualitative observations of Sylven[3] that the method of fixation greatly influences the mast cell count, a higher and more constant count being obtained when the lead-acetate-formalin fixative is used.

Comparative figures for histamine in homogenate of normal tissue are given in Table II.

Comparative figures for histamine content of inflamed tissue are unfortunately not available. The only figures known to the author are those obtained by Zon, et al.,[15] who found that normal rabbit skin contained an average of 9 μ gm. histamine per gm. *dry* weight and lesions caused by injection of turpentine contained an average of 21.7 μ gm. per gm. dry weight. Harris found that a 50 per cent loss in skin histamine occurred an hour after a burn to the skin. High concentrations, 500-1000 μ gm., are reported by Riley and co-

TABLE II

Histamine Concentration of Normal Tissue

Tissue	*Histamine μgm./gm. wet weight*	*Investigator*
Subcutaneous (Rat)	16.0	Riley and West[11]
Liver (Rat)	0.3	" " "
Liver Capsule (Ox)	40.0	" " "
Skin (Dog)	2.3—23	Graham & co-workers[12]
Lung (Dog)	9.0	" " " "
Liver (Dog)	12.0	" " " "
Heart (Dog)	3.4	" " " "
Muscle (Dog)	1.6	" " " "
Skin: whole (Human, Adult)	6.0	Harris[13]
Epidermus (Human, Adult)	23.0	"
Dermis (Human, Adult)	4.0	"
Skin (Human, Adult)	10.0	Nilzen [14]
Skin (Human, Adult)	20.0	Pellerat & Murat [14]

workers[10] to occur in tumors in various tissues of dogs.

Comparative figures for mast cell content are not available owing to the variety of fixatives used by investigators and the various methods of computing and recording the number of mast cells. Even so, it can be seen from the semi-quantitative assessments of the mast cell content of various tissues reported in the literature[4, 17, 18] that human gingival tissue is comparatively rich in mast cells.

The lack of direct correlation between numbers of mast cells and histamine content may be the result of the irregular distribution of mast cells. Histamine estimations were made on homogenate of tissue samples 0.2-1 gm. in weight but mast cells were counted on sections only 7μ thick, with, possibly, resultant discrepancy in sampling. Only the estimation of histamine content and mast cell numbers on adjacent serial sections or the counting of mast cells in a large number of sections from the same piece of tissue would overcome this sampling error.

SUMMARY

The role of mast cells and histamine in the body is considered together with their possible importance in gingival disease.

Mast cell numbers and histamine content of 18 biopsy specimens of inflamed gingival tissue were determined. The mean number of mast cells was 47 per sq.mm. connective tissue; the mean histamine concentration was 60 μ gm. per gm. wet weight total tissue.

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Illustrations from

Periodontal Considerations Associated with Reversible Acquired Prognathism

Reprinted

The October, 1957, issue of the JOURNAL OF PERIODONTOLOGY (Vol. 28, page 298) included a case report by Dr. Sidney S. Friedman, Jr., describing the treatment of a patient with prognathism. Unfortunately, the illustrations in this article were reproduced very poorly and as a result the article lost much of its value. We are therefore publishing Figures 2 and 4 again which illustrate the preoperative condition as well as the results of therapy after one year.

The JOURNAL OF PERIODONTOLOGY is printed on high quality paper and the engravings of the highest quality are ordered. While it is true that the illustrations did not have much contrast, these republished pictures should be better. We apologize to Dr. Friedman (and the readers) and we hope sincerely that all copies of the repeated illustrations will be acceptable.

Fig. 2. Acquired prognathic relation. Note formation of diastemas in the lower anterior region and loss of vertical dimension, function and esthetics.

Fig. 4. A functional, physiologic occlusal relationship has been established in centric and the various eccentric positions.

Centric Relation and the Postural Position of the Patient

by SANFORD C. FRUMKER, D.D.S.,* *Cleveland, Ohio*

DENTISTRY has always been interested in occlusion. However, it is only comparatively recently that enough has been known about the principles of occlusion to allow selective grinding to be used as a routine therapeutic procedure.

The pioneer work of Schuyler[5,6] elucidated many of the principles of occlusion. However, much of his work was primarily applicable to artificial teeth, and it took the efforts of Steven Brown[1] to work out methods and principles more applicable to the natural dentition. Since Brown presented his work there has been a tremendous upsurge in the recognition of the occlusal factor in periodontal disease, and a great deal is now written about occlusal adjustment by grinding of the natural dentition. Considerable attention in the literature has been paid to specific techniques for occlusal adjustment. However, little attention has been paid to the postural position of the patient at the time the adjustment is done.

Moyers[4] recent experiments have done much to elucidate the nature of centric relation. Goldman[2] describes centric relation as "a practiced, learned, neuromuscular pattern and a position of mandible to maxilla based on a neuromuscular pathway ranging from physiologic rest position to terminal tooth contact when a posterior, unstrained, hingelike movement of the mandible is effected." Should any part of a tooth deflect the mandible from this closure pattern, maximum intercuspation will occur in an eccentric position. The areas of the teeth causing this deviation of the mandible away from centric relation are called centric relation premature contacts.

Since centric relation is the position into which the mandible is most often directed, and since patients tend to swallow in centric relation, interferences in this position are the ones most likely to be operated by the patient and consequently most likely to result in occlusal traumatism. Understanding the role of the premature contact as an etiologic factor and the significance of its elimination, it becomes of great importance to know whether the location of this initial contact is dependent upon the postural position of the patient. The entire path of closure is not the question. What is important is whether the postural position of the patient has any effect on centric relation at the plane of initial tooth contact!

This question has not been widely discussed. Eberle[7] is quoted as advocating that "centric relation is taken in (a) supine relaxed position." Based on their recent electromyographic studies, the Shpuntoff's[8] "believe the most accurate condition for recording centric position . . . exists when the patient is standing erect."

MATERIAL AND METHOD

Fifty random patients were selected, both in the Western Reserve University Dental Clinic and the private office. Forty two of the patients were white, eight were negro. Thirty one were female, nineteen were male. The age range was from seventeen to fifty-four. Each patient had at least four upper posterior teeth occluding with at least four teeth in the lower arch.

By the use of Corning 30 gauge green wax and the jaw jiggling method,[3] centric relation was located for each of the patients. It was first recorded with the patient sitting upright. Then with the patient's head tilted backwards, and finally with the patient bending forward with his head between his legs. In both the upright and backward positions the head was supported by the headrest. After thirty-five patients it was decided to vary the experiment. The

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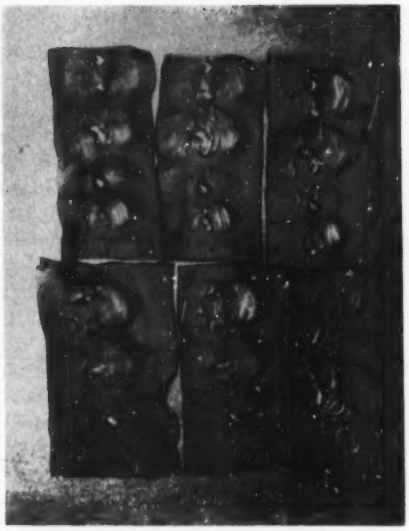

Fig. 1. 30 gauge wax registrations of centric relation prematurities on the left side of a 48 year old male patient. Reading left to right, top row, 1, patient sitting erect; 2, with head tilted back; 3, with head tilted forward. Bottom row, 4, erect with head unsupported; 5, patient standing; 6, right side of patient showing no contact. Note registrations are the same regardless of the position of the patient.

next five patients were recorded sitting upright and then standing. The next ten patients were recorded sitting upright with the head supported, then sitting upright with the head unsupported, and then standing.

RESULTS AND DISCUSSION

In all the patients, regardless of the postural positions, the wax recordings were the same. The areas of first contact in centric relation were in every one of the above cases at the same place on the same teeth. In some instances there were markings with distinctive configurations, and these were reproduced the same in all the positions of the patient.

It was noted that the position hardest to get an accurate reading in was with the patient's head between his legs. In this position there was a tendency for the patient to bite through the wax thus making the recording inaccurate. Contrarily, it was easiest to get the patient into centric relation with the head supported by the headrest and tilted backwards.

Since physiologic rest position is a postural position, by defining centric relation as encompassing the entire path from rest position to maximum occlusion, this automatically makes centric relation a postural position by definition. However, this is of little significance in occlusal adjustment, as the only part of centric relation we are interested in is that at the plane of initial tooth contact. Our object is to make initial tooth contact, centric occlusion, and maximum occlusion all one and the same. The centric relation of prime importance for occlusal adjustment is that at initial tooth contact, and the work herein applies only to centric relation as it occurs along this plane.

Although this work was done on patients with at least four pairs of contacting posterior teeth, there is no reason to conclude it should not apply as well to patients with less teeth, or to patients having removable partial prosthesis. However, it should not be construed as having any particular significance for the edentulous patient.

SUMMARY

1. Centric relation is of significance to occlusal adjustment by grinding primarily at the plane of initial tooth contact.

2. In patients with at least four pairs of contacting posterior teeth, regardless of the postural position of the head, and regardless of whether the head was supported or not, centric relation at the plane of initial tooth contact was the same.

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THE AMERICAN INSTITUTE OF DENTAL MEDICINE CASE HISTORY SERVICE

The Institute continues to offer Case Histories from the broad field of Dental Medicine for the year 1957-58. These will include original 35 mm. Kodachrome slides of the oral condition, pertinent laboratory findings, medical background, roentgenograms, photomicrographs, diagnostic criteria and whatever else may be indicated in each individual case, with a "general discussion" of the disease. A loose leaf binder and matching Kodachrome slide box will be furnished to each participant. The fiscal year of the Institute runs from October 1 to September 30.

This monthly service began October 1952 and has met with outstanding success. During these five years it has provided a total of 810 one-year subscriptions and the Histories were accompanied by 121 Kodachrome slides.

Anyone subscribing to this Case History Service is entitled to full membership in The American Institute of Dental Medicine. Members of the A.D.A. or A.M.A., or their foreign equivalent, are eligible to apply. Due to the great demand for this Service, it is recommended to subscribe at an early date for immediate delivery.

For further information, address all communications to the Secretary, Mrs. C. Novembri, 2240 Channing Way, Berkeley 4, California.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Two fellowships intended for graduate dentists interested in research and teaching careers in oral pathology and periodontology are now available. In addition to participation in an extensive research program, the fellowships offer training in oral pathology in a laboratory with an active biopsy diagnostic service, and an opportunity for preparing for advanced degrees. There is an annual stipend of $5,000 and the fellowships are renewable.

Additional information may be obtained from Dr. Irving Glickman, Department of Oral Pathology and Periodontology. 136 Harrison Avenue, Boston, Massachusetts.

Editorials • • • •

PROGRESS

A review of the activities of the American Academy of Periodontology for 1957 will convince anyone that progress is being made. The Spring Conference, held at the University of Michigan under the general supervision of Drs. William R. Mann, Donald A. Kerr, Sigurd Ramfjord, Joseph Barkley, James Robinson and Willa Yeretsky, was most noteworthy. This Conference deserves better support from the membership; the program for the 1958 Spring Conference is published elsewhere in this issue, and anyone interested is urged to attend.

The annual meeting in Miami Beach was also carefully developed and successfully staged by Secretary Dr. Clarke Chamberlain (and his efficient secretary, Mrs. Lorrie Hill), Dr. James Aiguier, Treasurer, with the assistance of course of the other officers and committees of the Academy. Particularly outstanding was the efficiency of the Local Arrangements Committee (Chairman, Roger Turner) and Drs. Nachazel (Chairman of the Scientific Sessions) Arnold Ariaudo (Chairman of the Program Committee) Robert Reeves (Chairman of the Clinic Committee) and their supporting committee members.

A third "project" of the Academy, the Journal of Periodontology completed another successful year. More pages and papers were published than ever before and the editor believes papers were of a high quality. A greater backlog of papers worthy of publication exists now than is desirable; a study is being made of methods of enabling more rapid publication.

These activities of the Academy, the Spring Conference, the Annual Meeting, and the Journal, are well established and will doubtless continue to grow. However, there are other valuable activities of this organization which deserve special note. The many committees of the Academy contribute much to the growth of the stature of the science of periodontology. Often they attract little attention, but particular mention should be made of the work of the Council of the Academy, the Membership Committee, Nomenclature Committee, Ad Hoc Committee, and the Research Committee. The 1957 committee members have been published; the 1958 list will be published as soon as it is available.

Another project worthy of attention is being organized by a special committee composed of Drs. Harold G. Ray, Chairman, James Aiguier, Willard C. Fleming, Maynard K. Hine, Donald Kerr and Robert Reeves. This committee has been active in obtaining funds to sponsor a workshop for teachers of periodontology to be held at the University of California School of Dentistry June 24, 25 and 26, 1958, and largely through the efforts of its chairman, has prepared an intensive program which should improve the teaching of periodontology in dental schools.

If all the committees of the American Academy of Periodontology continue to work as they have in the past couple of years, we are confident that the future of the Academy will be a bright one.

RESEARCH COMMITTEE REPORT COMMENDABLE

It has been customary to comment that there is little research being done in the field of periodontology, and yet figures presented in the report of the Research Committee of the American Academy of Periodontology, suggest otherwise. The Research Committee, composed of Drs. Harold Ray, Irving Glickman, and Sigurd Ramfjord, Chairman, reported that during the fiscal year July 1, 1956 to June 30, 1957, 41 projects directly related to periodontal conditions or diseases were supported to the amount of $550,323 from the United States Public Health Service alone. The committee stated that if all other studies of indirect and probable significance to periodontics were included, this figure would at least be doubled. For intramural activities in the National Institute of Dental Research about $130,000 is being spent annually on projects directly related to periodontics and another $100,000 on studies indirectly or partially related to this disease. If such studies as the chemistry of saliva or oral micro flora were included, this figure would be more than doubled. In addition to the above figures, the Army spent on periodontal research $41,376, the Navy $50,000 and the Air Force $45,000.

The Research Committee also reported two private companies supported periodontal research with an amount totaling $95,000.

This adds up to $1,011,699 on an annual basis for direct support of periodontal research, and if subjects related to periodontal conditions and diseases are included, the figure would probably exceed $2,000,000.

To all of this should be added the innumerable research studies carried on by dental schools and practitioners with no outside financial support. It is our hope that most of these research projects now under way will be carried to completion and eventual publication so that others may profit from the studies. Some of the investigations are basic in nature, while others concern improvements in therapy. This increased attack on problems associated with periodontal disease should prove fruitful—we hope!

EMORY UNIVERSITY SCHOOL OF DENTISTRY GRADUATE TEACHING FELLOWSHIP IN PERIODONTOLOGY

Emory University School of Dentistry offers a 24-month graduate teaching fellowship in Periodontology designed to qualify the participant for examination by the American Board of Periodontology and to give him a well-rounded background for the limited practice of Periodontology. This will begin on July 15, 1958.

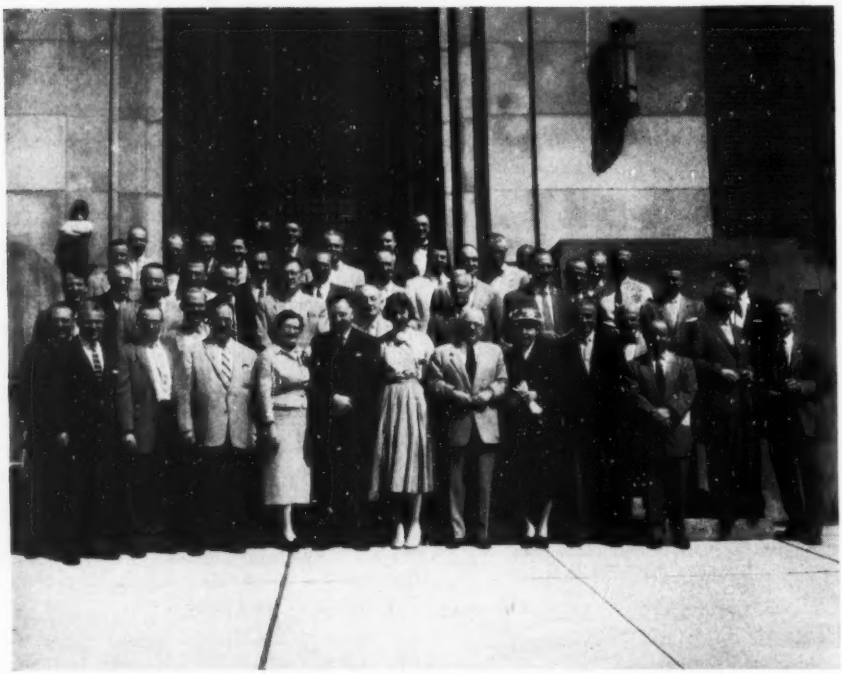
Requirements in the program include: advanced study in the field of basic biologic sciences including anatomy, bacteriology, histology, pathology, and physiology. Normal and abnormal occlusions will be studied from both an anatomical and the physiological points of view. Advanced theories and currently accepted treatment procedures will be evaluated and therapy in all periodontal branches will be experienced. Differential diagnosis or oral lesions, histopathology, and clinical experiences in oral pathology will consume a large part of the curriculum.

The fellow will be scheduled for clinical practice, didactic subjects of both lecture and seminar type teachings, and assigned readings. A thesis will be required for the student on an original research project accomplished throughout the two years study.

The fellow receives an annual stipend of $1,200. Appointments are made each year on April 1 for the succeeding 24-month period. Graduate dentists and senior dental students interested in obtaining further information should address their communications to Doctor Ernest L. Banks, Professor of Periodontology, Emory University School of Dentistry, 106 Forrest Avenue, N.E., Atlanta 3, Georgia.

Announcements • • • •

PARTICIPANTS IN THE SPRING SEMINAR, 1957, ANN ARBOR, MICHIGAN



(This photograph furnished by Dr. Stanley R. Suit from Western Reserve University)

One of the features of the activities of the American Academy of Periodontology is the Spring Seminar, held each year in Ann Arbor, Michigan. The program for the 1958 Seminar is announced elsewhere in this issue of the Journal. Since this is always an outstanding scientific meeting held in the pleasant environment at Ann Arbor, members and friends of the American Academy of Periodontology are urged to make plans to attend this year.

Details of the meeting may be obtained by writing Dr. Donald Kerr, University of Michigan School of Dentistry, Ann Arbor, Michigan.

SPRING CONFERENCE OF AMERICAN ACADEMY OF PERIODONTOLOGY

The Annual Spring Conference of the American Academy of Periodontology will be held April 21, 22 and 23, 1958, in the W. K. Kellogg Foundation Institute in the University of Michigan at Ann Arbor.

For further information write Dr. William R. Mann or Dr. Donald A. Kerr, University of Michigan School of Dentistry, Ann Arbor, Michigan.

The program is as follows:

Monday, April 21—8:00 a.m.-9:00 a.m.—Registration. Room 2004 Kellogg Building. 9:30 a.m.-12:00 noon—Physiology of Mastication. Robert E. Moyers, D.D.S., Ph.D., Professor of Dentistry, University of Michigan. 1:30 p.m.-4:00 p.m.—Diagnosis and Treatment of Facial Pain with Emphasis on the Temporomandibular Joint. Fred A. Henny, D.D.S., Chief, Division of Dentistry and Oral Surgery, Henry Ford Hospital, Detroit.

Tuesday, April 22—9:00 a.m.-12:00 noon—Muscular Changes Associated with Temporomandibular Joint Dysfunction. Harold T. Perry, Jr., D.D.S., Instructor in Orthodontics and Physiology, The Dental School, Northwestern University. 1:30 p.m.-4:00 p.m.—Emotional Factors in Facial Pain and Mandibular Dysfunction. Ruth Moulton, M.D., Assistant Clinical Professor of Psychiatry, Columbia University. 6:00 p.m.—Social Hour and Dinner; Michigan Union. Marcus L. Plant, M.A., J.D., Professor of Law and Faculty Representative to the Intercollegiate (Big Ten) Conference, University of Michigan.

Wednesday, April 23—9:00 a.m.-12:00 noon—Conclusions of the Temporomandibular Joint Clinic at Columbia. Laszlo Schwartz, D.D.S., Clinical Professor of Dentistry and Director, Division of Clinical Oral Physiology, School of Dental and Oral Surgery, Columbia University. 1:30 p.m.-2:00 p.m.—X-Ray Techniques of the Temporomandibular Joint. Albert G. Richards, M.S., Associate Professor of Dentistry, University of Michigan. 2:00 p.m.-3:30 p.m.—Panel Discussion: Temporomandibular Joint Disorders and their Treatment. Drs. Henny, Moulton, Moyers, Perry, and Schwartz. Moderator: Perry A. Ratcliff, D.D.S.

For further information write Dr. William Mann or Dr. Donald Kerr, University of Michigan.

ACADEMY WORKSHOP FOR TEACHERS JUNE 23-25

The American Academy of Periodontology is sponsoring a workshop for teachers of periodontology which will be presented at the School of Dentistry of the University of California in San Francisco on June 23, 24 and 25, 1958. It is planned that two members from each dental school in the United States and Canada who teach periodontics will be in attendance. The object of the workshop is to improve and correlate the teaching of periodontology in dental schools for the undergraduate. It is sponsored largely by Procter & Gamble Company with aid also from the American Academy of Periodontology, Lactona, Inc., Fund for Dental Education, and University of California.

Outline of the program is as follows:

June 23, 1958

A.M.—Introduction. Lecture: Periodontology in the Dental Curriculum. Lecture: Integration of Periodontology with Basic and other Clinical Sciences.

P.M.—Division of participants into the six following major committees and instruction in the workshop technique. Explanation of material to be covered. Assignment to quarters to begin work.

I. Techniques of Teaching in Periodontology.

II. Background Subject Matter Prerequisite to and Belonging within the Didactic Responsibility of Periodontology.

III. Subject Matter and Scope of Clinical Periodontics.

IV. School Clinical and Academic Procedures, Records and Audio Visual Aids. Curriculum time and sequence necessary to adequately teach periodontics within the confines of all fields in dentistry.

V. Teaching Prevention and Patient Responsibility. Consideration of epidemiology, and utilization of devices to educate the public through the profession by educating the undergraduate.

VI. Consideration of Research in Periodontology both Undergraduate and Graduate. A survey of research projects and needs directed toward teaching and clinical application.

Each committee will have a previously appointed leader and secretary who will moderate the committee discussions and stimulate the formation of conclusions.

Evening—Lectures: 1. Periodontal Disease and General Health. 2. Periodontology in the Undergraduate and Graduate Curriculum.

June 24, 1958

A.M.—Deliberation and library work of all committees.

P.M.—Demonstration lecture in didactic teaching in periodontology. Demonstration procedures in clinical instruction. Continued work of committees.

June 25, 1958

A.M.—Conference of entire body's opinion in didactic as well as clinical teaching. Panel—Leading teachers.

P.M.—Report of committees and proposals and recommendations. General discussion.

SOUTHWEST SOCIETY OF PERIODONTISTS

The third annual meeting of the Southwest Society of Periodontists will be held May 3 and 4, 1958 in the Roosevelt Hotel, Waco, Texas. Clinicians for the program will be Dr. Nate Friedman and Dr. Daniel Feder of Beverly Hills, California. They will discuss "Periodontal Prosthesis."

Ladies are invited and will be entertained.

For further information address Dr. James W. Clark, President, 303 West 8th Street, Texarkana, Texas or Dr. John H. Swindle, Jr., P. O. Box 3096 Westview Station, Waco, Texas.

THE UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

The University of Alabama School of Dentistry, Birmingham, Alabama, will offer the following refresher courses:

February 8, 9, 10, 1958

Periodontology for the General Practitioner will be given by Sumter S. Arnim, D.D.S., Ph.D., Professor of Pathology and director of the Postgraduate School of Dentistry in the Dental Branch of the University of Texas. Dr. Arnim has lectured widely on this subject; he will deliver a very practical type of program specifically designed for the busy general practitioner.

March 1, 2, 3, 1958

Occlusal Adjustment: Its relation to dental, periodontal and temporomandibular joint problems will be given by Sigurd Ramfjord, L.D.S., M.S., Ph.D., Associate Professor of Periodontics at the University of Michigan School of Dentistry. He has presented numerous courses and clinics on this subject in the United States and internationally.

Further information about these courses may be obtained by writing to the Director, Refresher Course Program, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

BETH ISRAEL HOSPITAL

The Dental Department of the Beth Israel Hospital announces a post-graduate course in Periodontia to be given in the Spring of 1958.

A two-week seminar in Periodontia is scheduled for March 17 to March 28. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00. A deposit of $150.00 is necessary for enrollment.

Co-Directors of the course are Henry M. Goldman, D.M.D. and Bernard Chaikin, D.M.D.

For further information, write to Director of Public Relations and Education, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Mass.

MEDICAL COLLEGE OF VIRGINIA SCHOOL OF DENTISTRY

Fellowships are available at the Medical College of Virginia, School of Dentistry for dental graduates to prepare for teaching and/or research. This program is supported in part by the National Institute of Dental Research and each fellowship carries with it a stipend of $5000.00 per annum.

Applicants may be accepted as candidates for M.S. or Ph.D. degrees. In order to obtain such degrees a trainee is required to major in a basic science. The latter may be in either anatomy, biochemistry, microbiology, pathology, pharmacology or physiology. The trainee may also select a clinical discipline to be carried along with the basic science major.

The Medical College of Virginia, School of Dentistry also announces postgraduate courses in Periodontics for the session beginning September 1958. These courses, both one and two years in length, are designed to prepare the interested dental graduate for specialty practice in Periodontics and to qualify applicants for admission to examination by the American Board of Periodontology.

Complete details may be obtained by addressing Dr. Holmes T. Knighton, Director, Graduate and Postgraduate Studies, Medical College of Virginia, School of Dentistry, Richmond 19, Virginia.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

The Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine announces a three-day refresher course (April 7, 8, 9, 1958) in Diagnosis and Management of Occlusal Disharmonies under the direction of Dr. W. George F. Schmidt, Professor of Clinical Dentistry, Northwestern University School of Dental Medicine. He will present a practical course devoted to the detection and correction of trauma from occlusion. It is a course for the general practitioner, the periodontist, orthodontist and prosthodontist. Technics for detecting occlusal disharmonies will be explained and demonstrated on patients. The technic for equilibrating the occlusion will be described in lectures, demonstrated on models and performed on patients.

For further information and application write to Director of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Massachusetts.

MARQUETTE UNIVERSITY SCHOOL OF DENTISTRY

Marquette University School of Dentistry will present refresher courses in Clinical Occlusion, with Course No. 1 being presented January 15, 16, 17, 18, 19. Cost $150.00. Repeat Course No. 1 will be held January 21, 22, 23, 24, 25, cost $150.00. Advanced Course No. 2 will be presented January 27 and 28, cost $60.00.

For further information write Division of Postgraduate Studies, Marquette University School of Dentistry, 604 N. 16th Street, Milwaukee, Wisconsin.